NEONATAL SEIZURES
A COMPREHENSIVE STUDY

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CERTIFICATE

This is to certify that this Dissertation titled **"NEONATAL SEIZURES – A COMPREHENSIVE STUDY"** is the bonafide original work of **Dr. J. SENTHILKUMAR** in partial fulfillment of the requirement for **MD (Branch VII)** Paediatrics examination of the Tamilnadu Dr.M.G.R Medical University to be held in April 2011.

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DECLARATION

I, **Dr. J. SENTHILKUMAR**, solemlnly declare that this dissertation "**NEONATAL SEIZURES – A COMPREHENSIVE STUDY**" is a bonafide record of work done by me in the Department of Paediatrics, Government Stanley Medical College and Hospital, Chennai.

This dissertation is submitted to the **Tamilnadu, Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of MD Degree (Paediatrics) **Branch - VII, Paediatrics** Examination to be held in April 2011.

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CONTENTS

INTRODUCTION

Seizures are the most distinctive manifestation of neurologic dysfunction in the newborn infant. Moreover, neonatal seizures often herald potentially devastating forms of brain injury. In earlier reports, seizures occurred in up to 3 in 1,000 full-term infants and up to 60 in 1,000 premature infants. However, the reported incidence of neonatal seizures varies widely across studies, a variability that is primarily the result of inconsistent diagnostic criteria, as well as the often subtle clinical manifestations of neonatal seizures, and their potential confusion with non epileptic neonatal behaviors. Regardless of their precise incidence, it is clear that seizures are more common in the newborn period than at any other time in life, and that the tendency toward recurrent seizures and status epilepticus is far greater in the newborn.

Neonatal seizure has an adverse effect on the neurodevelopment of the baby and may predispose to cognitive, behavioral or epileptic complication later in life. Hence it is indeed a real emergency to diagnose neonatal seizures as early as possible and treat it to prevent complications later in life.

REVIEW OF LITERATURE

PATHO PHYSIOLOGY

The decreased seizure threshold in the newborn reflects the developmental events active in the immature brain. In essence, the newborn brain has a transient overdevelopment of excitatory systems compared to inhibitory systems. For example, the immature brain has a transient over expression in the density of excitatory amino acid (primarily glutamate) receptors and a relative paucity of glutamate reuptake transporters. Together these features translate into more prolonged and intense contact of glutamate with postsynaptic receptors. Furthermore, these immature glutamate receptors are far more permissive of cationic influx, facilitating membrane depolarization and seizure activation. In contrast, inhibitory gamma-amino butyric acid (GABA) ion channels are relatively under expressed in the immature brain. In fact, in certain areas of the developing brain these immature GABA may be depolarizing (i.e., excitatory) rather than hyperpolarizing (i.e., inhibitory). In addition to these cellular factors, differential development of neural systems may enhance the excitatory state of the immature brain and predispose to seizures.

DIAGNOSIS OF NEONATAL SEIZURES

The clinical manifestations of neonatal seizures differ in many ways from those in older patients. The behavioral features of seizures in the newborn may be very subtle, in some cases confined to autonomic and subtle motor phenomena. In addition, the motor manifestations are often disorganized, and an orderly homunculus-based progression of convulsive activity (i.e., "Jacksonian march") is very uncommon.

The peculiar clinical characteristics of seizures in the newborn infant likely reflect the immature state of brain development. In late gestation and early postnatal life, active but incomplete developmental processes include cortical organization, axonal and dendritic branching, and the development of synaptic connections. Myelination commences around term but at this stage is largely confined to the deep subcortical regions of the brain. The relatively underdeveloped organization of the cortex and undermyelination of axons likely underlies the disorganized convulsive activity and lack of orderly seizure propagation in the newborn. For the same reasons, primary generalized seizures are very rare in the newborn. In accordance with the caudal-torostral gradient of brain development, the cortical development of the deep limbic system, including its connections to the diencephalic and brainstem structures, is relatively advanced compared to the more rostral neocortex. This fact may underlie the prevalence of behaviors referable to the limbic system, diencephalon, and brainstem, such as the sucking and chewing oromotor

automatisms, excessive drooling, occulomotor activity, and respiratory irregularities seen in subtle seizures.

Clinical diagnosis of neonatal seizures

Clinical seizure subtypes. Broadly speaking, clinical seizures may be defined as paroxysmal alterations of neurologic function, including behavioral, motor, and/or autonomic changes. First, non epileptic mimics of clinical seizures are common in the newborn. These seizure-like behavior patterns may occur in the normal newborn (e.g., non-nutritive sucking) and non epileptic paroxysmal clinical changes are common in encephalopathic newborns.

Given these diagnostic challenges, clinical seizure types may be categorized broadly into four groups: subtle seizures, clonic (focal, multifocal) seizures, tonic seizures, and myoclonic seizures. In many cases, more than one type of seizure occurs in a newborn over time.

Subtle seizures are the most common subtype, comprising about half of all seizures in term and premature newborns. Subtle seizures include a broad spectrum of behavioral phenomena, occurring in isolation or in combination. Ocular phenomena are common and include tonic eye deviation, roving "nystagmoid" eye movements, and sudden sustained eye opening with apparent visual fixation. Oro-bucco-lingual movements include chewing, sucking, or lip-smacking movements, and are often associated with a sudden increase in drooling. Various alternating limb movements ("progression movements") have been described, including pedaling, boxing, rowing, or swimming movements.

Autonomic phenomena, including sudden changes in skin color and capillary size, may occur alone or in combination with various motor manifestations. Such autonomic paroxysms are usually associated with initial tachycardia, and if sustained, with later bradycardia and possibly apnea. Uncommonly, and unlike clonic seizures, some cases of subtle seizures may be provoked or intensified by stimulation. Although the association between clinical and EEG events is variable, most subtle seizures are not associated with EEG seizures. Based on their inconsistent association with EEG seizures, as well as their poor response to conventional anticonvulsants, many consider these subtle seizures to be nonepileptic "brainstem release phenomena."

Clonic seizures are stereotypic and repetitive biphasic movements with a fast contraction phase and a slower relaxation phase. Clonic seizures may be unifocal, multifocal, or generalized. Clonic seizures that remain unifocal are usually not associated with loss of consciousness. The most common cause for clonic seizures that remain unifocal is neonatal stroke . Other causes of unifocal seizures include focal traumatic contusions, subarachnoid hemorrhage, or metabolic disturbances.

Tonic seizures have a sustained period (seconds) of muscle contraction without repetitive features. Tonic seizures may be generalized or focal. Generalized tonic seizures, which may closely mimic decerebrate or decorticate posturing, are most common in premature infants with diffuse neurologic dysfunction or major intraventricular hemorrhage (IVH). Generalized tonic

seizures are often associated with other motor automatisms or with clonic seizures, as well. Typically, infants are lethargic or obtunded between these seizures. Certain features suggest that these seizures may be nonepileptic in origin. Specifically, they may be precipitated by tactile or other stimuli, suggesting reflex discharges, and may be abolished by repositioning or light restraint. Finally, the clinical events are typically not associated with electrographic seizure patterns. The background EEG pattern tends to have multifocal or generalized voltage depression and undifferentiated frequencies, and, in some cases, a markedly abnormal burst suppression pattern. Overall, the prognosis of tonic seizures is very poor, except in some cases of post asphyxial seizures where an outcome may be less grim.

Myoclonic seizures are distinguished from clonic seizures by their lightning fast contractions and non rhythmic character. These seizures may occur in a multifocal or generalized pattern. Even when repetitive, myoclonic seizures tend to be irregular or erratic in nature. In some cases, myoclonic seizures may be elicited by tactile or auditory stimulation or suppressed by restraint. The electro-clinical association of myoclonic seizures is variable, and when present, the myoclonic contraction is usually associated with a single high-voltage spike and followed by a slow-wave complex. Typically, myoclonic seizures are associated with diffuse and usually serious brain dysfunction resulting from etiologies such as perinatal asphyxia, inborn errors of

metabolism, cerebral dysgenesis, or major brain trauma. Myoclonic seizures are usually associated with a poor long-term outcome.

Seizure mimics.

In the newborn it may be difficult to distinguish between normal immature behaviors (e.g., non-nutritive sucking), abnormal but non epileptic behaviors (e.g., "jitteriness"), and true epileptic manifestations. The following clinical guidelines may help distinguish true epileptic seizures from seizure mimics. These guidelines are most reliable with suspected clonic seizures but even then are not infallible.

First, true epileptic seizures are rarely stimulus-sensitive. Second, epileptic seizures cannot be abolished by passive restraint or repositioning of the infant. Third, epileptic seizures are often associated with autonomic changes or ocular phenomena. "Jitteriness" (tremor) may be distinguished from clonic seizures by the equal amplitude and faster equiphasic rhythm, compared to the slower, fast-and-slow components of clonic seizures. Generally, normal non epileptic behaviors are associated with a normal interictal examination. Conversely, abnormal but non epileptic repetitive behaviors often occur in encephalopathic infants with an abnormal interictal exam.

A temporal association between repetitive clinical events and simultaneous repetitive EEG changes is the strongest supportive evidence for true epileptic seizures. However, using electrographic monitoring to confirm the epileptic nature of suspicious clinical events is more complicated and

controversial in the newborn. This is particularly true when clinical seizure events are not accompanied by EEG changes, a situation most often seen with subtle seizures and generalized tonic seizures.

a) Epileptic apnea in the newborn. Apnea is not uncommon during neonatal seizures, but is rarely the only manifestation. Most infants with epileptic apnea will at some point in their course develop other seizure manifestations. Epileptic apnea may be difficult to distinguish from apnea due to other causes, such as neurologic depression, prematurity, sedative medications, and respiratory illness. However, there are several helpful distinguishing features. Neonatal epileptic apnea rarely lasts longer than 10 to 20 seconds. Bradycardia is often an early accompaniment of nonepileptic apnea, whereas in epileptic apnea, an initial tachycardia is more common, only followed in more prolonged seizures by later bradycardia. The EEG discharges that accompany epileptic apnea are often mono rhythmic (most commonly α frequency); in addition, they are usually focal over the temporal regions, suggesting an epileptogenic focus in the limbic system. Conversely, non epileptic apnea is not accompanied by EEG changes except for amplitude suppression that may develop during prolonged apnea.

b. Benign neonatal sleep myoclonus is a relatively common and sometimes dramatic non epileptic form of myoclonus. This condition presents in the first week of life, and resolves spontaneously (i.e., without treatment) over weeks to months. The convulsive activity emerges during quiet non rapid

eye movement (non-REM) sleep and is rapidly abolished by arousal. Myoclonic activity often builds up dramatically in both intensity and distribution over a period of minutes. Unlike other non epileptic behaviors, this form of myoclonus may be precipitated in some cases by gentle rhythmic rocking or tactile stimuli, and gentle restraint may actually increase rather than abolish the myoclonus. These events never occur during wakefulness and the neurologic examination is normal. Anticonvulsants are not indicated, and, in fact, benzodiazepines may exacerbate the myoclonic jerks. The long-term outcome is normal and later epilepsy does not develop

EEG diagnosis of neonatal seizures.

Ideally an EEG study should be recorded as soon as a seizure is first suspected, and preferably not later than 24 hours after. If such an EEG is normal, particularly if a suspected clinical event is captured during the EEG recording, then subsequent EEGs are only indicated if the clinical spells keep recurring. Whenever possible, several suspect events should be captured on EEG to confirm the true epileptic nature of the events. The absence of EEG changes during several clinical events, especially when the interictal EEG background is normal, is suggestive of a non epileptic process. If the initial EEG captures the features of seizure activity and antiepileptic drugs are started, a period of continuous video-EEG monitoring is recommended because anticonvulsant medications may abolish only the clinical manifestations, allowing ongoing and undetected EEG seizures to persist. Ideally, EEG

monitoring should continue for 24 to 48 hours after the last recorded electrographic seizure. A repeat EEG after 1 week may have particular prognostic value. The need for subsequent EEG studies as a guide to discontinuation of anticonvulsant medications is controversial.

Etiologic diagnosis of neonatal seizures.

At the first suspicion of neonatal seizures, the immediate focus should be the exclusion of rapidly correctable and potentially injurious processes, including hypoglycemia, hypocalcemia, and hypomagnesemia, among others. After seizures are confirmed and management has commenced, the etiology should be pursued through a rational and orderly approach, with a stepwise interpretation of the facts, and refocusing of the diagnostic plan. The evaluation should start with a careful history of pregnancy, labor and delivery, and family, followed by a detailed clinical examination for signs of dysmorphism, trauma, skin lesions, and unusual odors. The neurologic examination should include a careful and accurate clinical description of the seizure features, the infant's mental status, and cranial nerve examination as well as interictal movements, muscle tone, and deep tendon and primitive reflexes. Certain clinical signs may suggest specific etiologies and may facilitate a more rapid etiologic diagnosis. Next, selected special diagnostic techniques may be necessary to pursue or confirm the etiology of seizures, including **blood studies, cerebrospinal fluid (CSF) analysis, EEG recording, and neuroimaging studies.** Using such an

orderly and rational approach, most neonatal seizure etiologies should be identifiable

1.Hypoxic-ischemic encephalopathy:

The leading cause of neonatal seizures is cerebral hypoxia-ischemia, which may occur in the antenatal, intra partum, or neonatal periods. Perinatal asphyxia is implicated in 25% to 40% of neonatal seizures. Post asphyxial seizures occur in infants with moderate-to-severe grades of encephalopathy, that is, with obtundation, stupor, or coma. In addition, these infants tend to have muscle hypotonia, altered deep tendon reflexes and, in severe cases, brainstem abnormalities. Intrapartum asphyxia should never be a diagnosis of exclusion, and should satisfy certain criteria, including evidence of significant fetal distress, immediate postnatal "depression" at birth, and subsequent altered mental status. Commonly accepted criteria for immediate neonatal depression include an Apgar score of <5 at 5 minutes of life.

2. Focal ischemic injury:

i. Neonatal arterial stroke occurs in around 1 in 4,000 live births. In most cases, the etiology of neonatal strokes remains unknown Seizures are the most common presentation of stroke in the newborn period, and stroke is the second most common cause of neonatal seizures, accounting for 15% to 20% of cases.

ii. Cerebral vein thrombosis usually occurs in the large dural sinuses, particularly the posterior aspects of the superior sagittal sinus. Although the

presentation of cerebral vein thrombosis may be subtle, with lethargy often the only feature, approximately 60% of cases develop neonatal seizures.

3. Intracranial hemorrhage:

Intracranial hemorrhage is implicated in approximately 10% of neonatal seizures. The location of hemorrhage and the clinical features of the seizures varies with gestational age. With term infants, post hemorrhagic seizures are most commonly associated with primary subarachnoid hemorrhage and less often with subdural hemorrhage (SDH). Primary subarachnoid hemorrhage occurs more frequently after difficult prolonged or traumatic labor, including forceps and vacuum deliveries. However, primary subarachnoid hemorrhage may occur after apparently uncomplicated labor (i.e., so-called parturitional hemorrhage). Infants with seizures associated with primary subarachnoid hemorrhage have a good long-term outcome in 90% of cases. About half of all subdural hemorrhages (SDH) diagnosed in the newborn are complicated by seizures, usually presenting in the first days of life.

Post hemorrhagic seizures in the preterm infant have different features and a more ominous prognosis. These seizures are usually associated with severe IVH, or its parenchymal complication, periventricular hemorrhagic infarction (PVHI). Seizures following severe IVH usually present within the first 3 days of life in sick, very premature infants.

4. Central nervous system infections:

Central nervous system infections from a variety of agents, including viral, bacterial, or other organisms such as toxoplasmosis, may have neonatal seizures as a prominent part of their presentation. The mechanism of seizures in central nervous system infections may be through direct cerebritis or vaso-occlusive injury with secondary seizures. The onset of infection-related seizures obviously depends on the various organisms and onset of infection. Of the bacterial infections, meningitis due to Group B streptococcus and Escherichia coli are the most common, and in these cases, seizures usually develop in the latter part of the first week or later.

5. Metabolic disturbances:

Two types of metabolic disturbances may result in neonatal seizures: (i) transient and rapidly correctable disturbances, and (ii) inherited and usually persistent causes.

i. Transient metabolic disturbances include disturbances of blood glucose and electrolyte disturbances such as hypoglycemia, hypocalcaemia and hypomagnesaemia.

a) Hypoglycemia is especially common in infants with intrauterine growth retardation, diabetic mothers, or perinatal asphyxia. Less commonly, hypoglycemia may be a prominent feature of certain inborn errors of metabolism (e.g., galactosemia, glycogen storage diseases) or

hyperinsulinemic conditions (e.g., Beckwith-Wiedeman syndrome, nesidioblastosis).

b) Hypocalcaemia accounts for approximately 3% of neonatal seizures. Currently, hypocalcaemic seizures are usually associated with perinatal asphyxia or endocrinopathies due to maternal neonatal hypoparathyroidism or deletion syndromes of chromosome 22, including the DeGeorge syndrome.

ii. Inborn errors of metabolism are an uncommon cause of neonatal seizures; nevertheless, neonatal seizures have been described in a long list of such conditions. Certain of these conditions are more likely to be associated with seizures, including nonketotic hyperglycinemia, pyridoxine dependency, sulfate oxidase deficiency, glutaric aciduria type II, and urea cycle defects. The most common diagnostic abnormalities associated with these conditions include metabolic acidosis, hyperammonemia, hypoglycemia and ketosis.

6. Cerebral dysgenesis:

A number of dysgenetic cerebral lesions may be associated with neonatal seizures. In many, but not all, cases these lesions can be demonstrated in vivo by CT or MRI scan. Conditions most commonly associated with neonatal seizures are disorders of neuronal migration (e.g., heterotopias, lissencephalies) or disorders of neuronal organization (e.g., polymicrogyria).

7. Epileptic syndromes in the newborn infant

a) Benign familial neonatal seizures

b) Benign idiopathic neonatal seizures

c) Neonatal myoclonic encephalopathy (NME)

d) Ohtahara syndrome

e) Migrating partial seizures of infancy (Coppola syndrome)

TREATMENT OF NEONATAL SEIZURES

1. **Reversing rapidly correctable causes**
2. **Specific anticonvulsant agents**

Hypoglycemia: Even when other primary etiologies are identified for seizures, hypoglycemia should be excluded or corrected as shown below.

Hypocalcaemia and hypomagnesaemia: Even if hypocalcaemia seizures respond to antiepileptic medications, the low calcium levels should be corrected. Infants treated for hypocalcaemia should also receive magnesium because calcium administration increases renal magnesium excretion, and magnesium administration increases serum calcium levels. Of note, magnesium administration may result in transient weakness and hypotonia, even with normal serum levels.

Once the diagnosis of seizures is strongly suspected or confirmed, **anticonvulsant agents like Phenobarbital** should be started. The administration of these agents should occur with careful cardiorespiratory monitoring.

PROTOCOL FOR THE MANAGEMENT OF NEONATAL SEIZURES

Stabilize vital functions

↓

Correct transient metabolic disturbances

Hypoglycemia (target blood sugar 70-120 mg/dL)

10% dextrose water IV bolus dose 2 mL/kg followed by a continuous infusion at 8 mg/kg/min

↓

Hypocalcemia - 5% calcium gluconate IV at 4 mL/kg (need cardiac monitoring)

↓

Hypomagnesemia - 50% magnesium sulfate IM at 0.2 mL/kg

↓

Phenobarbital 20 mg/kg IV load (1mg/kg/min)

Cardiorespiratory monitoring

↓

5mg/kg IV (may repeat to total dose of 40 mg/kg)

↓

Consider intubation / ventilation

↓

Lorazepam 0.05 mg/kg IV (may repeat to total dose of 0.1 mg/kg)

↓

Phenytoin 20 mg/kg slow IV load

↓

5 mg/kg IV (may repeat to total dose of 30 mg/kg)

↓

Pyridoxine 50-100 mg/kg IV (with EEG monitoring)

Shah GS et al [34], B P Koirala institute of Health Sciences, Nepal studied 99 neonates with neonatal seizures. They found out the incidence was 10.3/1000 live births. The seizures were common in male babies. Birth asphyxia

(44%), the most common cause observed followed by meningitis and septicemia (22%). Among metabolic abnormalities hypoglycemia was found in 20 (22%) and hypocalcaemia in 10 (11%). In birth asphyxia, the most common type of seizure observed was subtle seizures 20 (50%) followed by focal clonic 10 (25%) and multi focal clonic 5 (12.5%). Tonic type of seizures was observed in 7.5% and myoclonic in 2 (5%).

Ajay kumar et al, Maulana Azad Medical College, New Delhi [11], enrolled 90 babies into the study over one year period. Overall incidence was 1.17% (0.69% in term babies and 6.14% in preterm babies). The majority of babies are preterm very low birth weight developed seizures before 5 days of life. Perinatal asphyxia was responsible in 44.44% babies followed by metabolic abnormalities (23.33%). Fifty two babies (57.8%) developed seizure within 48 hours of life, out of which 20 babies had seizures in less than 12 hours of life. He found LBW as an important risk factor, as clinical seizures in them often indicate severe brain injury and are associated with serious morbidity. The risk of neonatal seizures varies inversely with birth weight. As reported by lanska et al, the incidence of seizures in VLBW babies was 10.14% and 0.59% in normal weight babies. In this study seizures types observed are multifocal clonic seizres (42.24%) followed by generalized tonic (21.55%) , subtle (8.19%), focal clonic (6.47%) and myoclonic (0.86%). However, Mizrahi and Kellaway and Scher et al have reported subtle seizures as the most common type of neonatal seizures in their studies.

Carrascosa et al., of Hospital General, Espana [26] studied the incidence, etiology and course of neonatal convulsions in the Albacete Health District between 1991 and 1993. He found the incidence of 1.4/1000 in full term live births and 13.4/1000 preterm live births. Etiology: hypoxic-ischaemic encephalopathy 32%, malformations or cerebral dysgenesis 24%, intracranial hemorrhage 16%, with less frequency: infections, metabolic and pharmacological changes 8%, epileptogenic diagnosis 4%. COURSE: 10 of the RN (40%) died, 8 (32%) had sequelae, although in 3 cases these were transient, and 7 had developed normally (28%).

Amar et al of Mahatma Gandhi Institute of Medical Sciences, Wardha, Maharastra [33] studied the incidence, etiological factor, days of onset, clinical types and various biochemical abnormalities in neonatal seizures. 110 neonates with neonatal seizures who were delivered at their hospital and developed seizures before 28 days of life are admitted.

The incidence of neonatal seizures was 16.69/ 1000 live births. The seizures were more common in male babies. 64 (58.2%) neonates were born to primigravida mothers while 46 (41.8%) neonates were born to multiparous women. In term babies (n=4650), 77 neonates had seizures out of that 24(31.1%) had subtle seizure, 36 (46.7%) had clonic seizure, 15 (19.4%) had tonic seizures and 2 (2.5%) had myoclonic seizures. In birth asphyxia (n=47), the most common type of seizures observed were subtle seizure 15(31.9%), followed by focal clonic 12(25.5%) and multifocal clonic 10(21.2%). Among

metabolic abnormalities, early hypocalcaemia was seen in 6 (46.1%) while late hypocalcaemia were detected in 7 (53.8%) babies. Hypoglycemia was more commonly seen after 3 days of life in 6 babies while in hypomagnesaemia 2 had seizures in first 2 days of life. Out of 110 babies, 21 (19.1%) neonates expired while 89 (80.9%) neonates were discharged. The most common causes of neonatal deaths were severe birth asphyxia seen in 9(42.8) neonates followed by IVH in 5 (23.8%), septicemia in 4(23.8%) and meningitis in 3 (14.2%) neonates.

The commonest cause of seizure in term babies was birth asphyxia with majority presenting to us within the first 72hrs. IVH and sepsis contribute maximum to seizure in preterm. Subtle seizures were the commonest type of seizure observed in term and preterm neonates.

Tinuade ogunlesi et al., [37] of Nigeria conducted a prospective study of consecutive newborn babies with seizures admitted to a Nigerian hospital between January and December 2006 to determine the risk factors for mortality in neonatal seizures.

Seventy-eight babies were studied. Thirty-six of these (46.1%) had seizures within the first 24 hours of life. The mean age at onset of seizure was 85.4 ± 106.1 hours. The leading etiologies included hypocalcaemia (65.4%), hypoxic-ischemic

encephalopathy (HIE) (60.3%) and hypoglycemia (50.0%). Severe anemia occurred in 56.4% of babies. Most (85.9%) had multiple etiologies while no

etiology was identified in 5.1%.The mortality rate was 43.6%. Significant risk factors for mortality included duration of seizure longer than 24 hours (p = 0.019), hypoglycemia (p = 0.001) and severe anemia (p = 0.004). The co-existence of HIE with hypoglycemia and hypocalcaemia was also more significantly associated with mortality (p = 0.03) than each of hypoglycemia and hypocalcaemia co-existing with HIE separately.

Rima M. Saliba et al., [39] of Texas evaluated risk factors for neonatal seizures. 207 **in**fants were diagnosed with cl**in**ical **neonatal seizures**. **In**formation was obta**in**ed from the **in**fant's birth certificate to assess the relation between **seizures** and birth weight, gender, ethnicity, place of birth, mother's age, method of delivery, parity, and multiple births. These factors were evaluated by univariate and multivariate analysis us**in**g logistic regression. For preterm **in**fants, a birth weight of <1,500 g was the strongest risk factor (relative risk (RR) = 9.1, 95% confidence **in**terval (CI): 4.7, 17.5), followed by birth **in** a private/university hospital (RR = 2.8, 95% CI: 1.5, 5.0) and male gender (RR = 1.8, 95% CI: 1.0, 3.4). For term **in**fants, significant risk factors **in**cluded birth by cesarean section (RR = 2.2, 95% CI: 1.5, 3.2), small birth weight for gestational age (RR = 1.9, 95% CI: 1.2, 2.9), birth **in** a private/university hospital (RR = 1.8, 95% CI: 1.1, 3.0), and maternal age of 18–24 compared with 25–29 years (RR = 1.6, 95% CI: 1.1, 2.3). Birth by assisted vag**in**al delivery and primiparity were marg**in**ally significant for term **in**fants. Birth weight is a significant risk factor for **neonatal seizures.**

Raj D Sheth et al of wisconson medical school, conducted a study to examine the influence of gestational age on seizures in the neonatal intensive care unit. A cohort of 4165 neonates admitted to a university intensive care unit between 1986 and 1995. The incidence, time of onset, and etiology of neonatal seizures in the cohort were distributed by gestational age.

Seizures occurred in 356 (8.6%) infants. The seizure rate was parabolically related to gestational age, such that infants at 30 to 36 weeks' gestation had a 4.8% rate compared with 11.9% and 14.1% rates for infants <30 and >36 weeks, respectively ($p < 0.001$). Seizures manifested earlier in infants <30 weeks and >36 weeks gestational age compared with neonates 30 to 36 weeks. Intraventricular hemorrhage was the principal etiology underlying the higher seizure rate for infants <30 weeks. Hypoxic-ischemic encephalopathy and congenital malformations were primary factors for infants >36 weeks ($p < 0.01$). Nervous system infections were evenly distributed across gestational age. Thus gestational age exerts a considerable influence on the incidence, onset, and etiology of neonatal seizures.

Sanjeev Kumar et al., [40] of SMGS hospital, Jammu conducted a study in all neonates < 28days of life who were hospitalized in the neonatal division, to assess the prevalence of neonatal seizures. The prevalence of Neonatal seizures in hospitalized neonates is 19.2%. There were 70.5% (72) males and 29.5% (30) females with a Male : Female ratio of 2.4:1. Majority of the neonates (45.09%) had seizures during first 24hrs. followed by 25.49% &

20.6% during next 24hr - 72hr and 72hr - 7days respectively. Majority of neonates (67.65) had history of birth asphyxia followed by 12.75% neonates who had no obvious clinical cause for seizures at admission. Septicemia alone, meningitis, kernicterus

and intracranial bleed each accounted for 11.76%, 3.92%, 2.94% and 0.98% respectively at admission. Majority 37.25% (38) neonates had multifocal clonic seizures followed by 26.47%, 23.52%, 12.74% subtle, tonic and focal clonic seizures respectively .None of the neonate had myoclonic seizures.

Tudehope et al., [35] Queensland, Australia had done the clinical spectrum and outcome of neonatal convulsions within an obstetric hospital population and were reviewed for the 5 years, 1978-82, inclusive. There were 156 convulsing neonates managed at the Mater Mothers Hospital (110 inborn, 46 outborn). The incidence of early neonatal convulsions for inborn babies was 3.0/1000 live births. Antenatal and perinatal risk factors were compared between the 156 convulsing infants and the 36,082 infants born during the same period who did not convulse. **The leading risk factors for convulsions were prematurity, intra-uterine growth retardation, low 5 min Apgar score, pre-ecclampsia, antepartum hemorrhage, twin pregnancy and breech presentation.** The predominant seizure type was tonic in 28.6%,

multifocal clonic in 27.2%, subtle in 18.4%, myoclonic in 15.0% and focal clonic in 8.8%. Mortality (31%) and long-term disability (43%) rates were high.

In Am J Epidemiol. 2001 Jan 15;153(2):103-7., an article published by Wen SW et al, Canada on Comparison of maternal and infant outcomes between vacuum extraction and forceps deliveries. Of all the births, 31,015 were delivered by vacuum extraction, and 18,727 were delivered by forceps. Compared with delivery by forceps, the adjusted risk ratios for third-/fourth-degree perineal laceration, intracranial hemorrhage, subdural or cerebral hemorrhage, intraventricular hemorrhage, subarachnoid hemorrhage, cephalhematoma, and neonatal in-hospital death were 0.48 (95% confidence interval: 0.45, 0.50), 1.28 (95% confidence interval: 0.73, 2.25), 0.97 (95% confidence interval: 0.49, 1.93), 0.99 (95% confidence interval: 0.16, 5.97), 5.44 (confidence interval: 1.26, 23.43), 2.02 (95% confidence interval: 1.89, 2.16), and 0.93 (95% confidence interval: 0.32, 2.70), respectively. The authors conclude that vacuum extraction causes less maternal trauma but may increase the risk of cephalhematoma and certain types of intracranial hemorrhage (e.g., subarachnoid hemorrhage).

Maheswari MC et al., [36] AIIMS, New Delhi, had done a comparative prospective survey on forceps delivery as a risk factor in epilepsy. **Three-hundred and eighty-one children born with forceps delivery and 372 with**

normal delivery were followed up for 4-7 years. More children in the forceps group developed seizures than in normal group, i.e. 22:10.

Pradhan et al., [37] studied the short term outcome of breech delivery at term in terms of perinatal mortality, Apgar scores, admission to the neonatal unit, birth trauma and neonatal convulsions. Of 1433 singleton term infants in breech presentation at onset of labour, 881 (61.5%) were delivered vaginally or by caesarean section in labour and 552 (38.5%) were born by prelabour caesarean section. There were three (0.3%) non-malformed perinatal deaths among infants born by vaginal delivery or caesarean section in labour compared with none in the prelabour caesarean section cohort. Compared with infants born by prelabour caesarean section, those delivered vaginally or by caesarean section in labour were significantly more likely to have low 5-minute Apgar scores and require admission to the neonatal unit.

Volpe JJ et al., [1, 12] took a retrospective review of 150 newborns with seizures evaluated at the same medical center and by the same investigator(s) from 1982 to 1987. The aims of the study were to determine in this current population the distribution of clinical seizure types, the distribution of causative etiologies, and the relation of etiology to the timing of onset of the seizures. Seizures were classified as subtle, multifocal clonic, generalized tonic, focal

clonic, and myoclonic. Subtle (65% of total) and multifocal clonic seizures (54% of total) were the most common seizure types. Subtle seizures usually occurred in combination with other seizure types. Only one seizure type was related to gestational age, i.e., focal clonic seizures in the term infant. Hypoxic-ischemic encephalopathy (65% of total) was by far the most common etiology in both preterm and term infants. Seizures with hypoxic-ischemic encephalopathy occurred characteristically early in the neonatal period, i.e., 90 percent in the first 2 days of life. Moreover, 80 percent of all seizures in the first 2 days of life were related to hypoxic-ischemic encephalopathy.

OBJECTIVES OF THE STUDY

1. To study the incidence of neonatal seizures.
2. To analyze the baseline characteristics like sex, birth weight, gestational age, intra uterine nutritional status and the mode of deliveries.
3. To study the clinical profile of neonatal seizures like etiology, various types, time of onset of seizures and bio chemical abnormalities.
4. To study the immediate outcome of neonatal seizures.

- Study period : Aug 2009 to Aug 2010
- Study design : Prospective study, Descriptive study
- Study place : NICU, RSRM hospital.
- All neonates delivered and developed seizures within 28 days of life were enrolled in this study. 132 babies developed clinical seizures over this study period. In all neonates enrolled in our study, the following information outlined below was collected and recorded.

(a) Baseline characteristics:

- These included sex, birth weight, gestational age and intra uterine growth status. Gestational age of all neonates was assessed using the modified Dubowitz or new Ballard scoring and classified as preterm (less than 37 completed weeks (259 days)), term (37 to 416/7 weeks (260 – 294 days)) and post term (42 weeks (295 days) or more). With the birth weight and gestational age, intra uterine status of these babies were categorized into appropriate for gestational age (AGA), small for gestational age (SGA), and large for gestational age (LGA) using reference charts (ref. cloherty).

b) Clinical profile of Seizures:

Each seizure episode reported by mother and subsequently observed by doctor in duty was recorded and relevant information was gathered like time of

onset of seizures (<24 hrs, 24 – 72 hrs, >72 hrs), and type of seizures. Seizures were classified according to Volpe's classification into Subtle, focal clonic, multifocal clonic, tonic and myoclonic seizures.

c) Determination of etiology :

Relevant clinical information was collected and recorded. Antenatal history of infections including TORCH, PIH and perinatal history of hemorrhage, chorio-amnionitis, fetal distress like meconeum stained liquor was recorded. Mode of delivery, history of birth asphyxia, details of resuscitation, apgar scores were recorded. All babies were examined daily, including standard neurological examination till they were discharged.

d) Investigations :

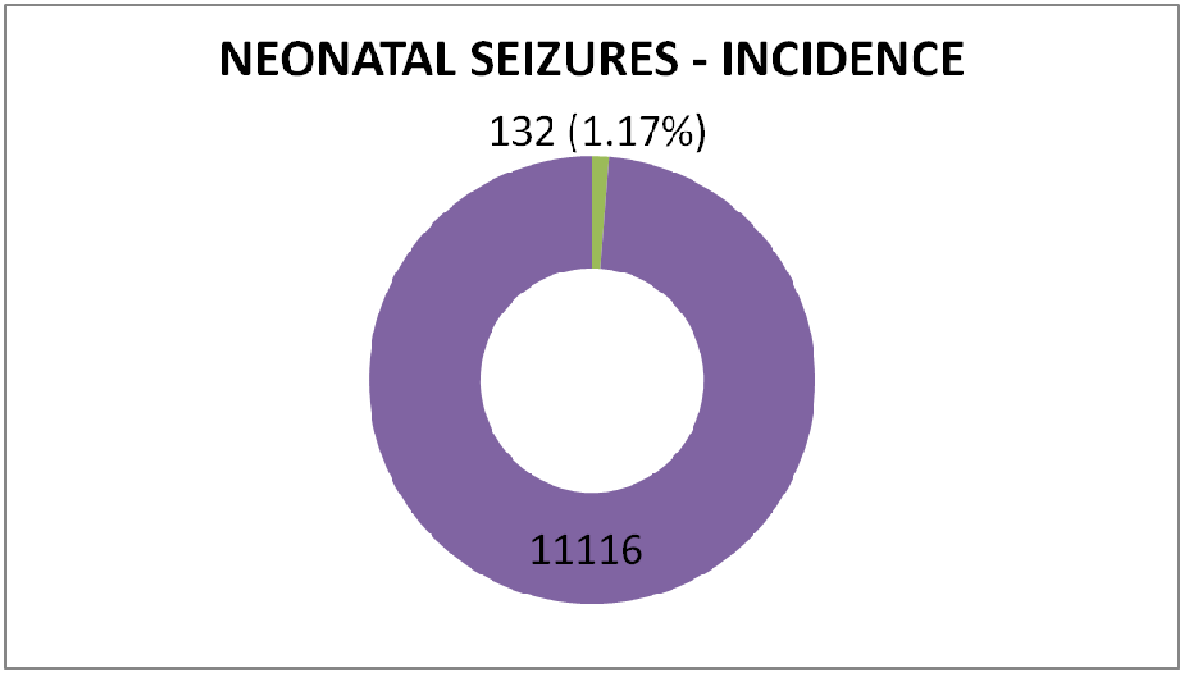
Essential investigations done in all the subjects including hemoglobin (Hb), packed cell volume (PCV), total count (TC), differential count (DC), platelets, blood glucose, serum calcium, C-reactive protein (CRP), blood for NEC, CSF analysis and urine for metabolic screening. Ultra sound cranium and EEG were done as early as possible. CT scan if there was any abnormal ultrasound, serum magnesium if there any refractory hypocalcaemia. All babies were treated as per the standard treatment protocol.

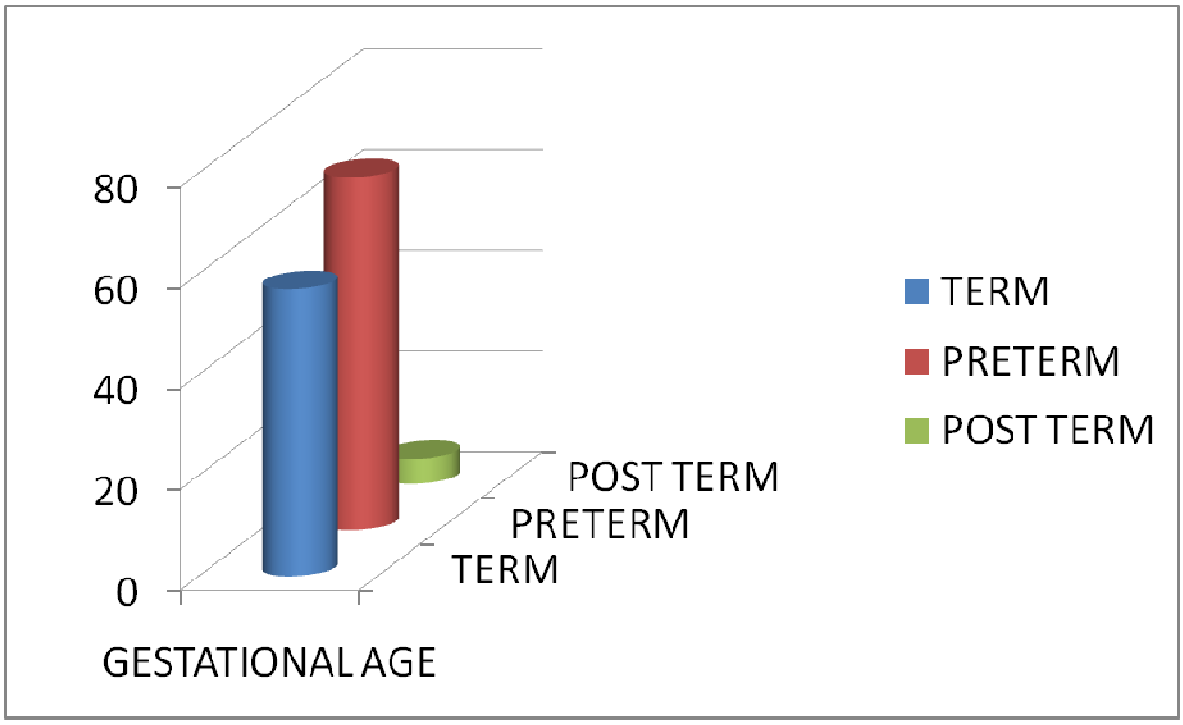
Various parameters like incidence, sex, birth weight, gestational age, intra uterine nutrition, mode of delivery, clinical type of seizure, time of onset, etiology and immediate outcome were analysed. Statistical analysis was done using Epi info software.

INCIDENCE OF NEONATAL SEIZURES

Of 11,248 live births delivered in RSRM during the study period, 132 neonates presented with neonatal seizures (11.7/1000 live births). 0.58% of term babies, 5.18% of preterm babies and 2.8% of post term babies were having neonatal seizures. Of all 2986 admissions in NICU per year, 4.4% of babies were neonatal seizures.

GESTATIONAL AGE	**NO. OF LIVE BIRTHS (n=11,248)**	**NEONATAL SEIZURES (n=132)**
TERM	9726	57 (0.58)
PRE TERM	1349	70 (5.18)
POST TERM	173	05 (2.8)


NEONATAL SEIZURES - INCIDENCE
132 (1.17%)
11116


80
60
40
20
0
POST TERM
PRETERM
TERM
GESTATIONAL AGE
TERM
PRETERM
POST TERM

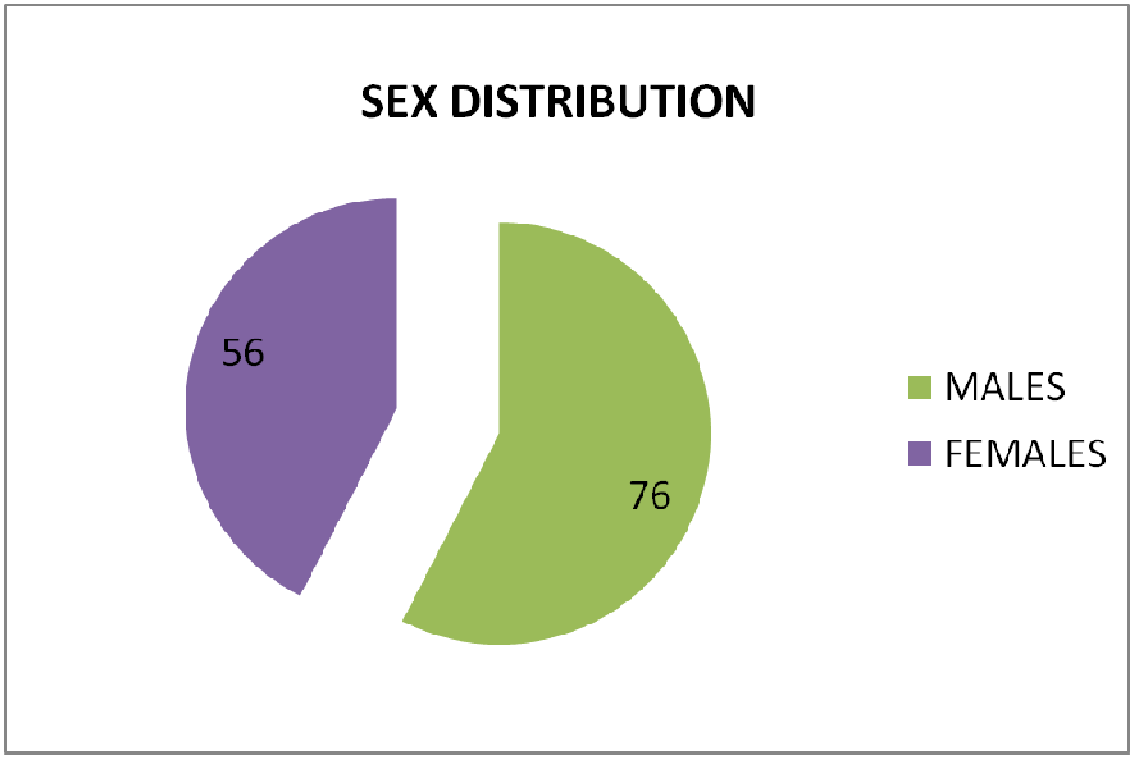
SEX DISTRIBUTION

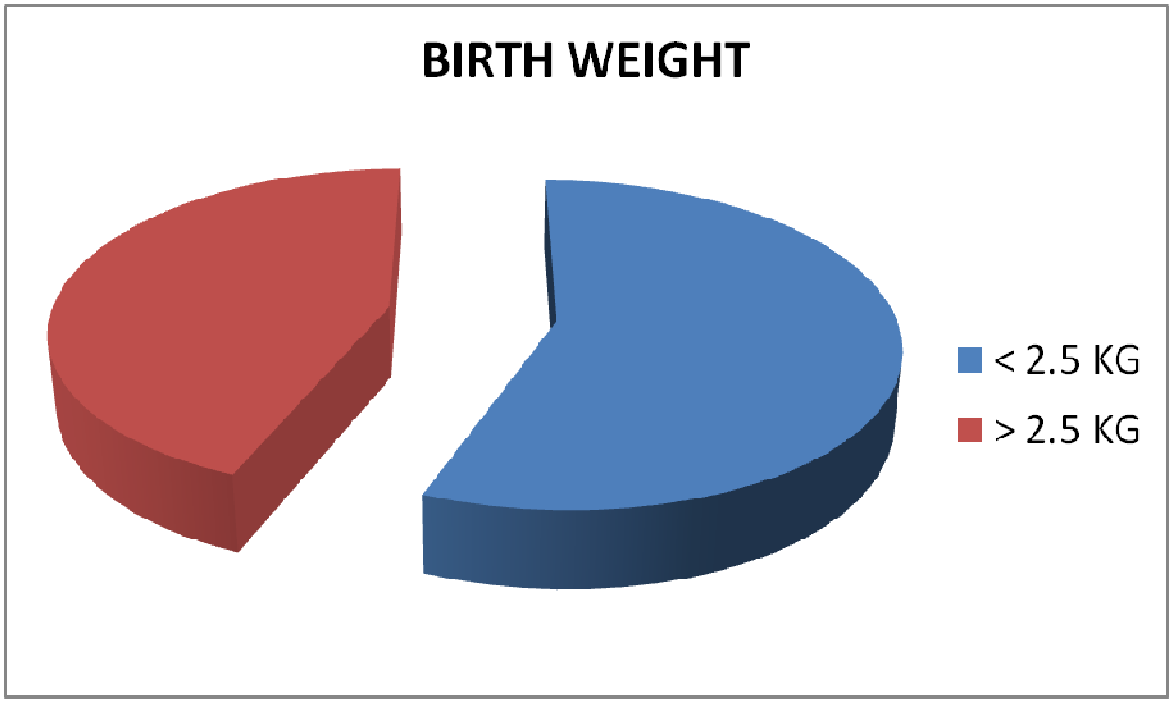
Of 132 babies studied 76 (57.5%) were males and 56 (42.5%) were females in the ratio of 1.4: 1, with male preponderance.

BIRTH WEIGHT

74 (56.1%) were low birth weight babies and 58 (43.9%) were having birth weight >2.5 Kg. when compared to overall live births 2.9% of LBW babies and 0.7% of babies of birth weight >2.5 Kg developed seizures.

BIRTH WT.	OVERALL DELIVERIES (n=11,248)	SEIZURES (n=132)
< 2.5 Kg	2500	74 (2.9)
> 2.5 Kg	8748	58 (0.7)


SEX DISTRIBUTION
56
76
MALES
FEMALES


BIRTH WEIGHT
< 2.5 KG
> 2.5 KG

OUTCOME OF NEONATAL SEIZURES

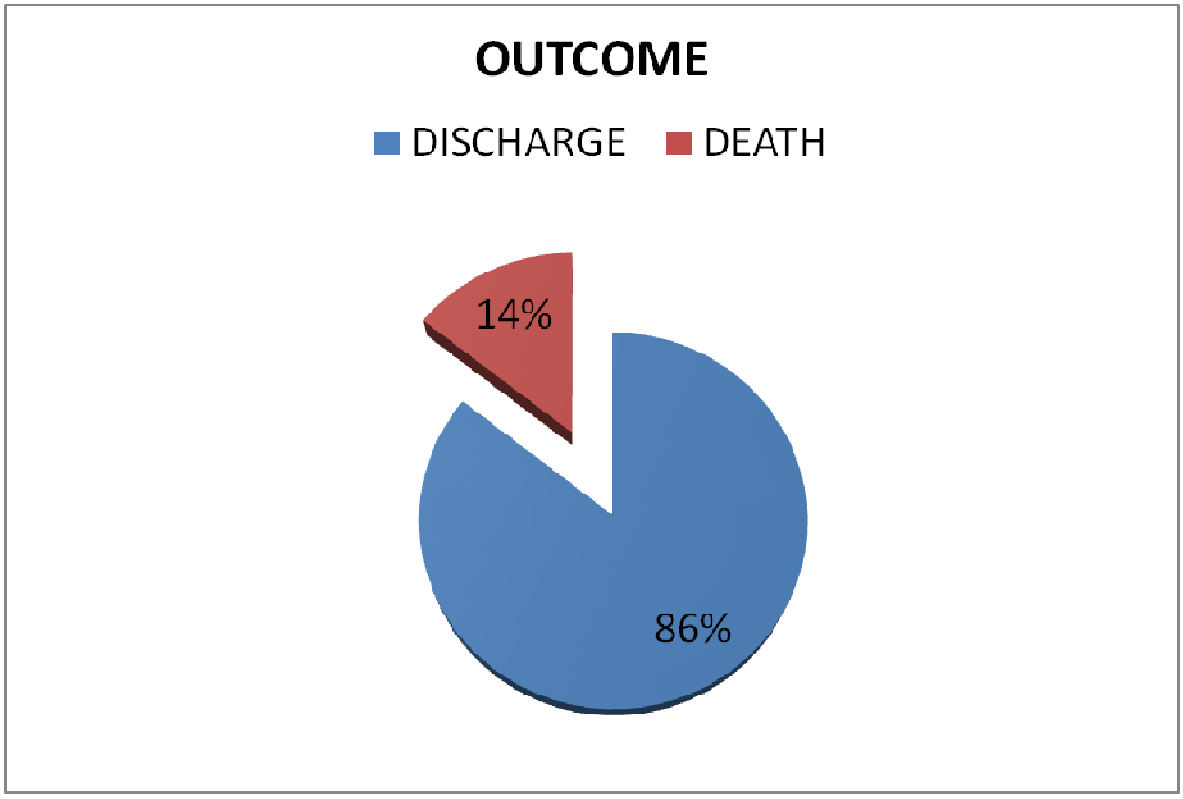
OUTCOME	SEIZURES (N=132)
DISCHARGE	113 (85.6)
DEATH	19 (14.4)

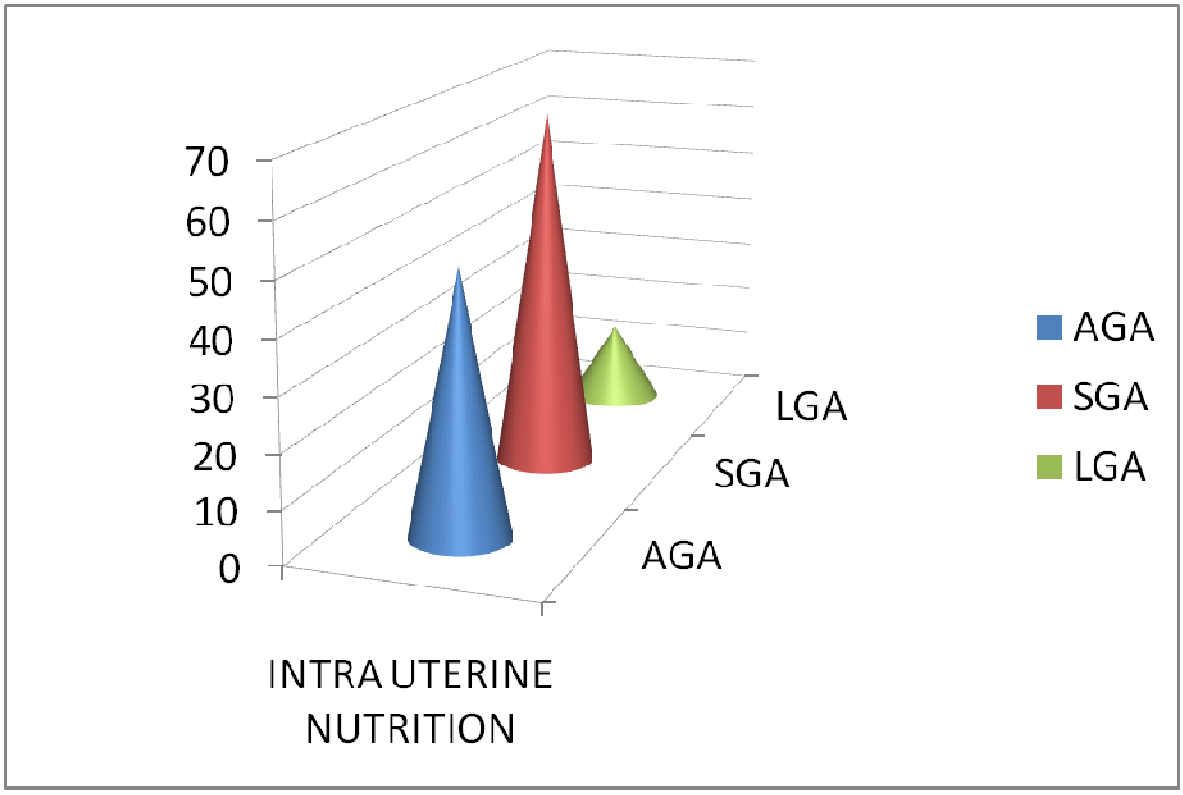
Of 132 babies 19 (14.4%) died and 113 (85.6%) discharged to home.

INTRA UTERINE NUTRITIONAL STATUS

It was found that 49 (37.1%) babies were AGA, 68 (51.5%) babies were SGA, 15 (11.4%) were LGA. Neonatal seizures found to be more prevalent in SGA babies.

NUTRITIONAL STATUS	SEIZURES (n=132)	% (100)
AGA	49	37.1
SGA	68	51.5
LGA	15	11.4


OUTCOME
DISCHARGE
DEATH
14%
86%

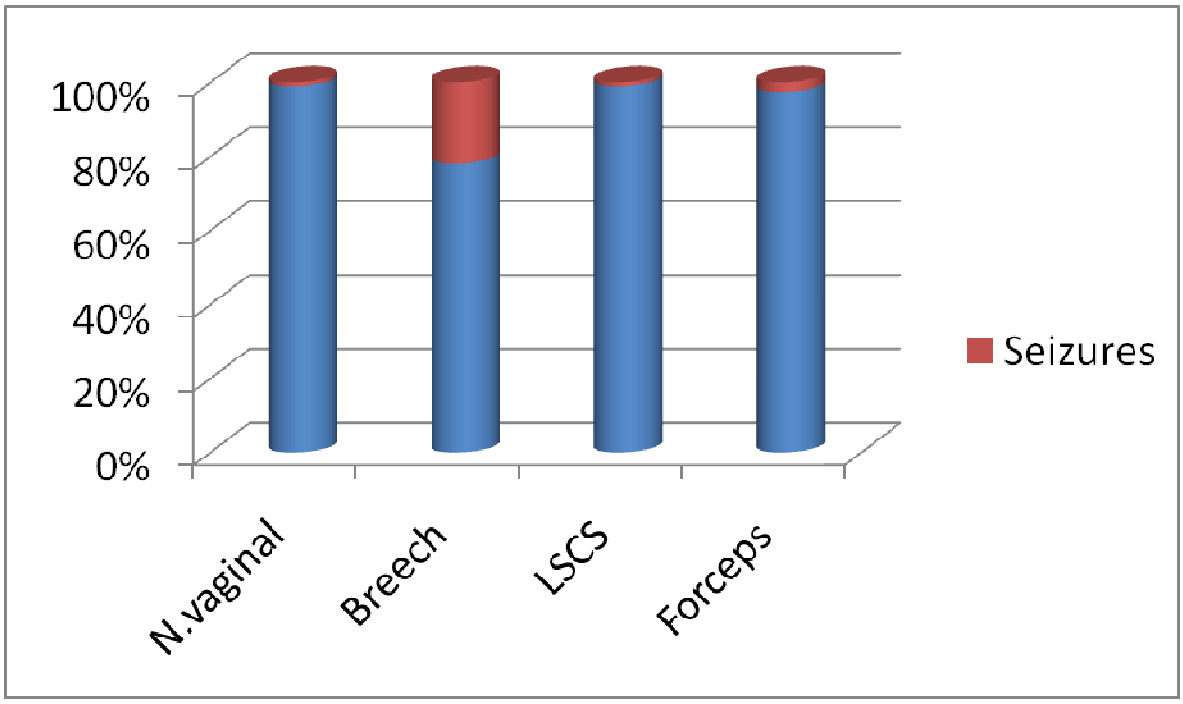

70
60
50
40
30
20
10
0
LGA
SGA
AGA
INTRA UTERINE
NUTRITION
AGA
SGA
LGA

MODE OF DELIVERY

MODE OF DELIVERY	SEIZURES (N=132)	OVERALL LIVEBIRTHS (N=11248)
VAGINAL DELIVERY	66 (50%)	6184 (1.1)
BREECH	7 (5.3%)	25 (28)
LSCS	49 (37.1%)	4658 (1.1)
FORCEPS	10 (7.6%)	381 (2.6)

1.1% of babies delivered vaginally, 1.1% of babies delivered by LSCS, 28% of babies delivered by breech and 2.6% of babies delivered by forceps developed neonatal seizures.

MODE OF DELIVERIES



NEONATAL SEIZURES IN TWINS

	LIVE BIRTHS (n=11,248)	**SEIZURES (n=132)**
SINGLE BORN	11,098 (97%)	128 (1.2%)
TWINS	150 (3.0%)	4 (2.7%)

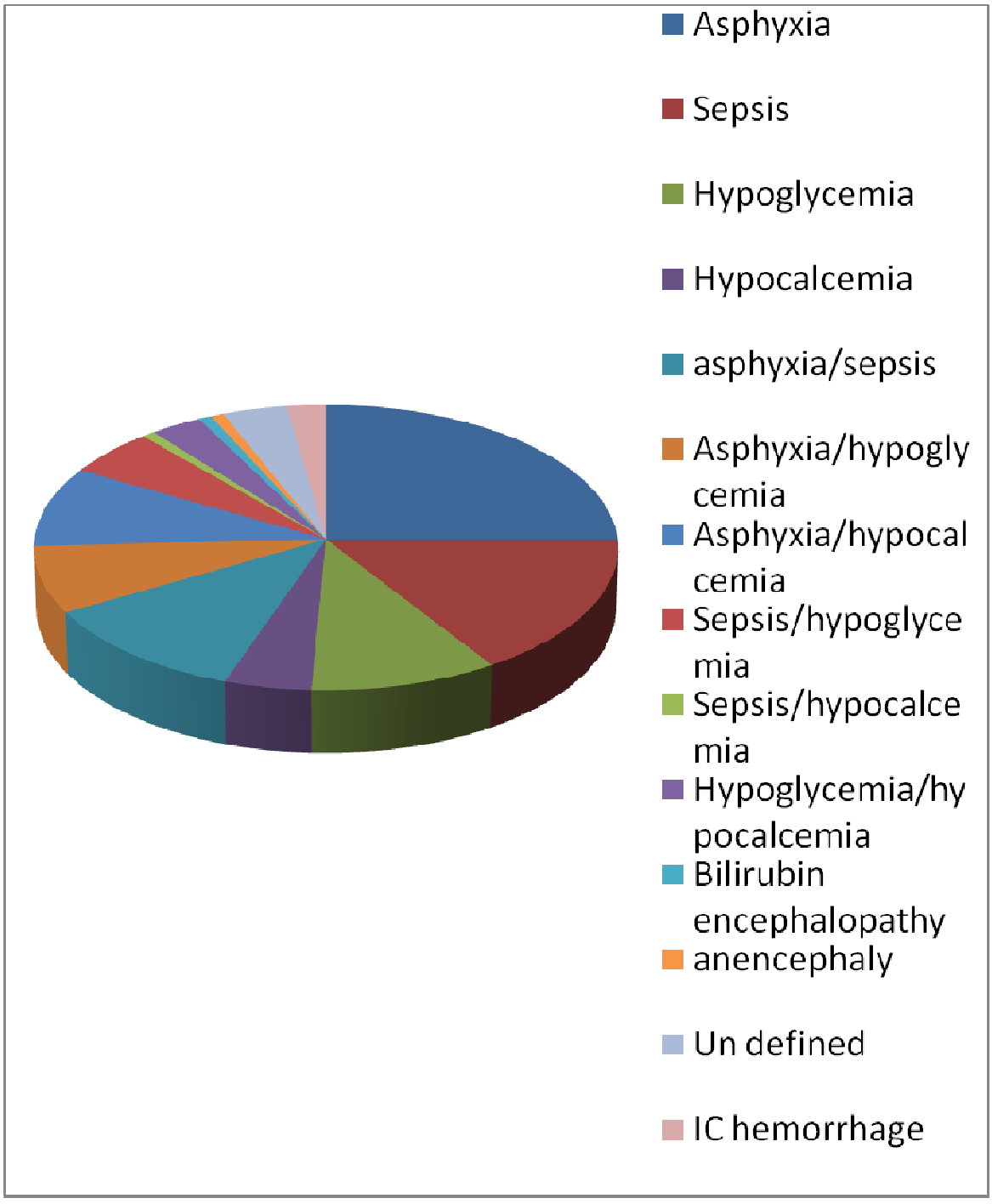
Of 132 babies 128 were delivered as singleton babies and 4 babies born as twins, which was about 1.2% and 2.7% of total singleton and twin deliveries respectively.

ETIOLOGY OF NEONATAL SEIZURES

CAUSES	INCIDENCE
BIRTH ASPHYXIA	70 (53%)
SEPTICEMIA	44 (33.3%)
HYPOGLYCEMIA	35 (26.5%)
HYPOCALCEMIA	24 (18.2%)
INTRA CRANIAL HEMORRAGE	03 (2.3%)
BILIRUBIN ENCEPHALOPATHY	1 (0.8%)
ANENCEPHALY	1 (0.8%)
UN KNOWN	5 (3.9%)

Birth asphyxia contributes to about 53% of cases, followed by sepsis 33.3%, hypoglycemia 26.5% and hypocalcaemia 18.2%. Cause remains unknown in 3% of cases.

ETIOLOGY OF SEIZURES



TYPES OF SEIZURES

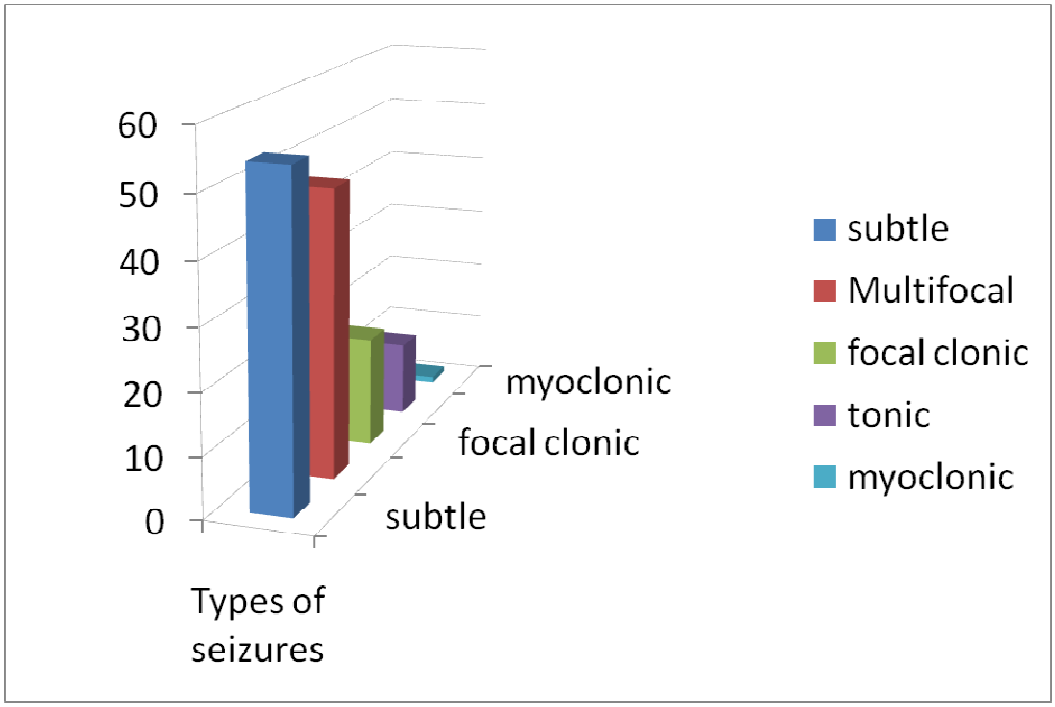
SEIZURE TYPE	n=132 (%)
SUBTLE	54 (40.9)
MULTIFOCAL	47 (35.6)
FOCAL CLONIC	18 (13.6)
TONIC	12 (9.1)
MYOCLONIC	1 (0.8)

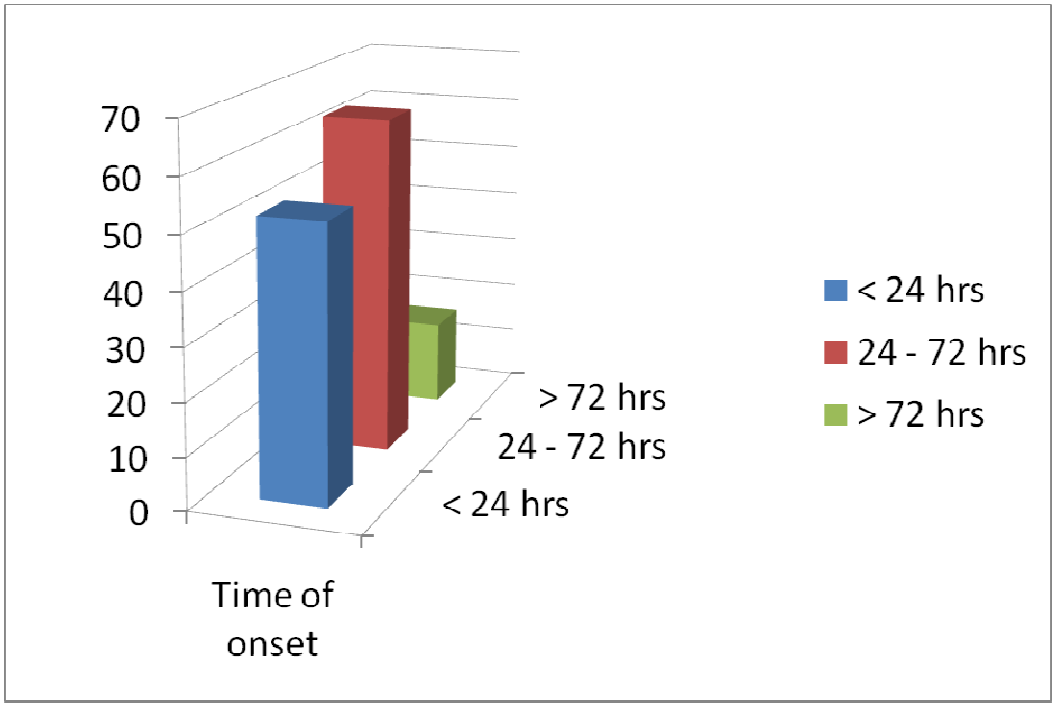
Subtle seizures (40.9%) were the commonest of all seizures, closely followed by multifocal clonic (35.6%).

TIME OF ONSET OF SEIZURES

TIME	SEIZURES n=132 (%)
< 24 HRS	52 (39.4)
24 – 72 HRS	64 (48.5)
> 72 HRS	16 (12.1)

In 64 (48.5%) babies seizure occur between 24 and 72 hrs whereas 52 (39.4%) babies had their seizure in < 24 hrs.


60
50
40
30
20
10
0
myoclonic
focal clonic
subtle
Types of seizures
subtle
Multifocal
focal clonic
tonic
myoclonic

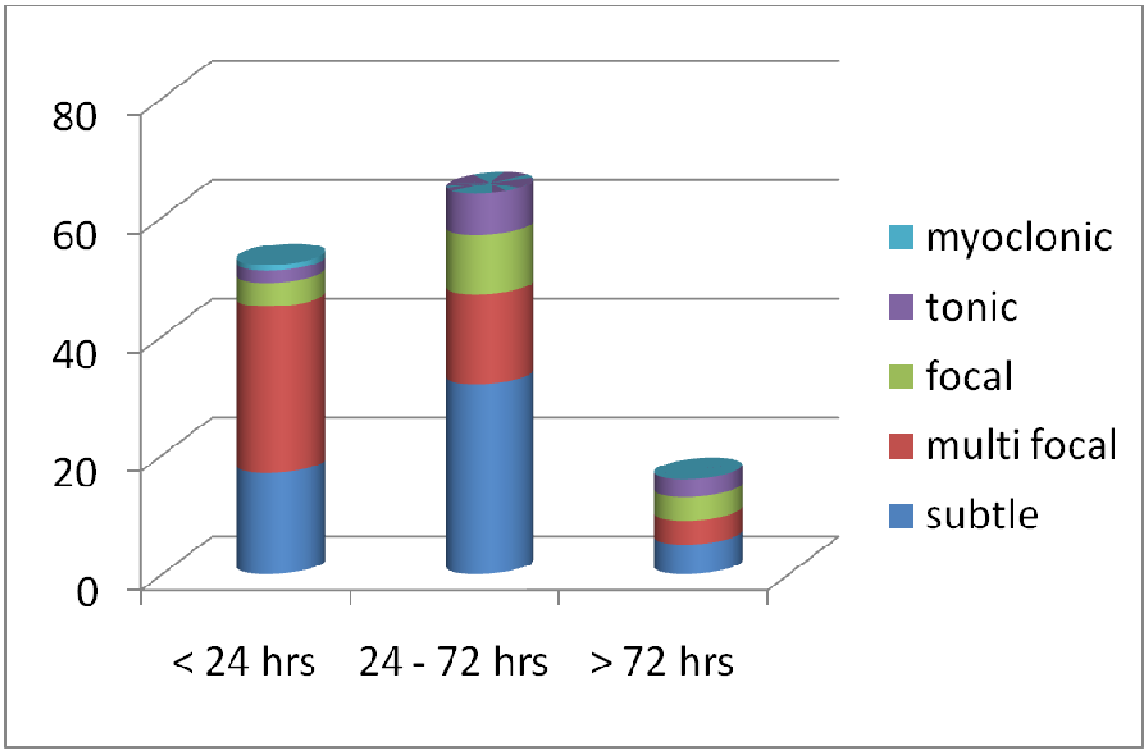

70
60
50
40
30
20
10
0
> 72 hrs
24 - 72 hrs
< 24 hrs
Time of onset
< 24 hrs
24 - 72 hrs
> 72 hrs

TYPES OF SEIZURES IN VARIOUS TIMES OF ONSET

TYPES	< 24 HRS (n = 52)	24 – 72 HRS (n = 64)	>72 HRS (n = 16)
Subtle (n=54)	17 (32.7)(31.5)	32 (50)(59.3)	5 (31.3)(9.3)
Multifocal clonic (n=47)	28 (53.8)(59.6)	15 (23.4)(31.9)	4 (25)(8.5)
Focal clonic (n=18)	4 (7.7)(22.2)	10 (15.6)(55.6)	4 (25)(22.2)
Tonic (n=12)	2 (3.8)(16.7)	7 (10.9)(58.3)	3 (18.8)(25)
Myoclonic (n=1)	1 (1(100)	0	0
( ) – 1- vertical percentage ( ) – 2 – horizontal percentage			

Multifocal seizures (53.8%) were common in < 24 hrs while subtle seizures (50%) were common in 24 – 72 hrs, which is **statistically significant (p-0.0295)**

TYPES OF SEIZURES IN VARIOUS TIMES OF ONSET

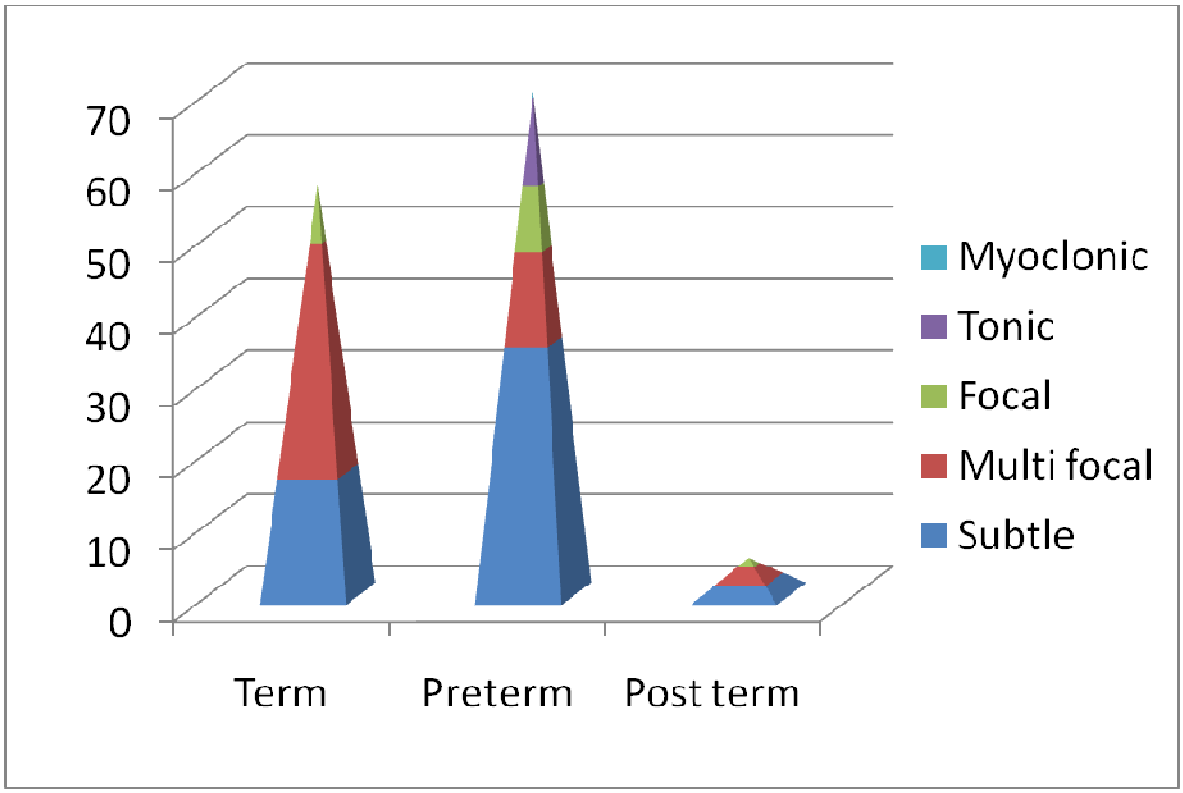


GESTATIONAL AGE WISE DISTRIBUTION OF TYPES OF SEIZURES

TYPE	TERM (n=57)	PRE TERM (n=70)	POST TERM (n=5)
Subtle (n=54)	17 (29.8)(31.5)	35 (50.0)**(64.8)**	02 (40.0)(3.7)
Multifocal clonic (n=47)	32 (56.1)**(68.1)**	13 (18.6)(27.6)	02 (40.0)(4.3)
Focal clonic (n=18)	08 (14.0)(44.5)	09 (12.9)(50.0)	01 (20.0)(5.6)
Tonic (n=12)	00	12 (17.1)(100)	00
Myoclonic (n=1)	00	01 (1.4)(100)	00
( ) – 1- vertical percentage ( ) – 2 – horizontal percentage			

Multifocal clonic seizures (68.1%) were common in term babies while subtle seizures (64.8%) were common among preterm babies, which is **statistically significant (p – 0.0006).**

GESTATIONAL AGEWISE DISTRIBUTION OF TYPES OF SEIZURES

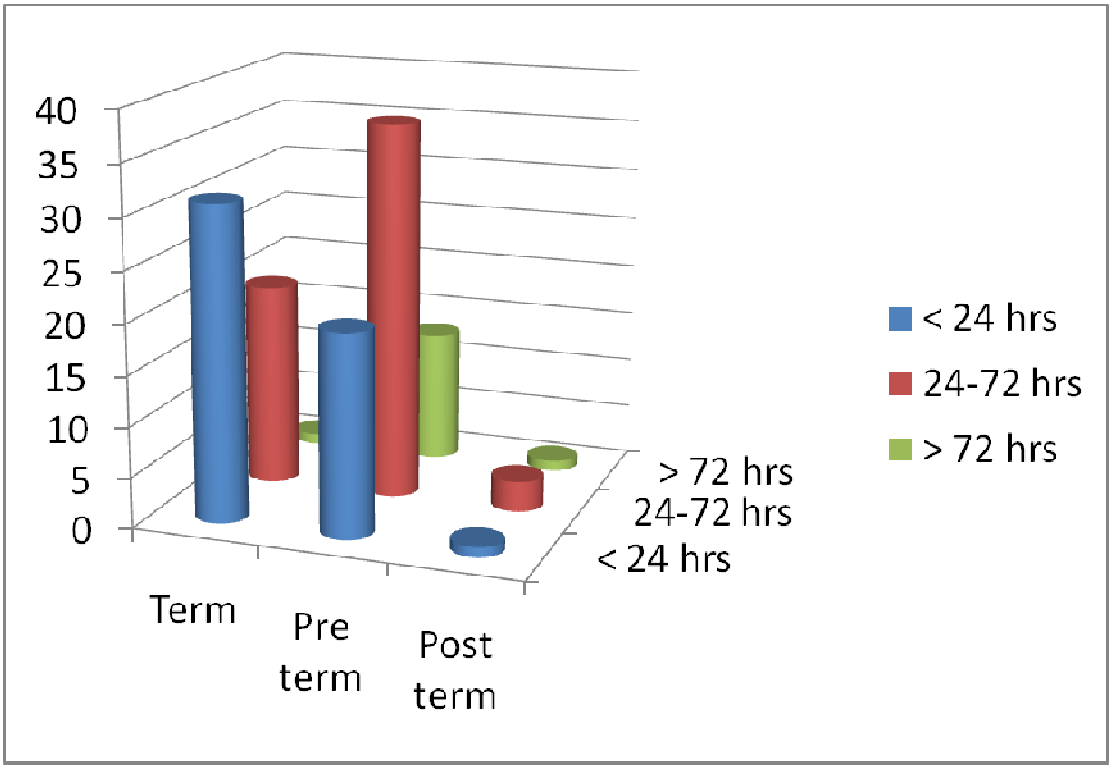


GESTATIONAL AGE AND TIME OF ONSET OF SEIZURES

GEST. AGE	< 24 HRS	24 - 72 HRS	> 72 HRS	TOTAL
TERM	**31**	24	2	57
Row %	**54.4**	42.1	3.5	100
Col %	59.6	37.5	12.5	43.2
PRETERM	20	**37**	13	70
Row %	28.6	**52.9**	18.6	100
Col %	38.5	57.8	81.3	53
POST TERM	1	3	1	5
Row %	20	60	20	100
Col %	1.9	4.7	6.3	3.8
TOTAL	52	64	16	132
Row %	39.4	48.5	12.1	100
Col %	100	100	100	100

In term babies 54.4% (31) babies developed seizures in <24 hrs whereas in preterm babies 52.9% (37) developed seizures between 24-72 hrs with the **significant p value of 0.012**.

GESTATIONAL AGE AND TIME OF ONSET OF SEIZURES



DISTRIBUTION OF NEONATAL SEIZURES IN INTRA UTERINE NUTRITION

Gestational age	AGA	SGA	LGA	TOTAL
TERM	19	**28**	10	57
Row %	33.3	**49.1**	17.5	100
Col %	38.8	41.2	66.7	43.2
PRETERM	27	**38**	5	70
Row %	38.6	**54.3**	7.1	100
Col %	55.1	55.9	33.3	53
POST TERM	3	2	0	5
Row %	60	40	0	100
Col %	6.1	2.9	0	3.8
TOTAL	49	68	15	132
Row %	37.1	51.5	11.4	100
Col %	100	100	100	100

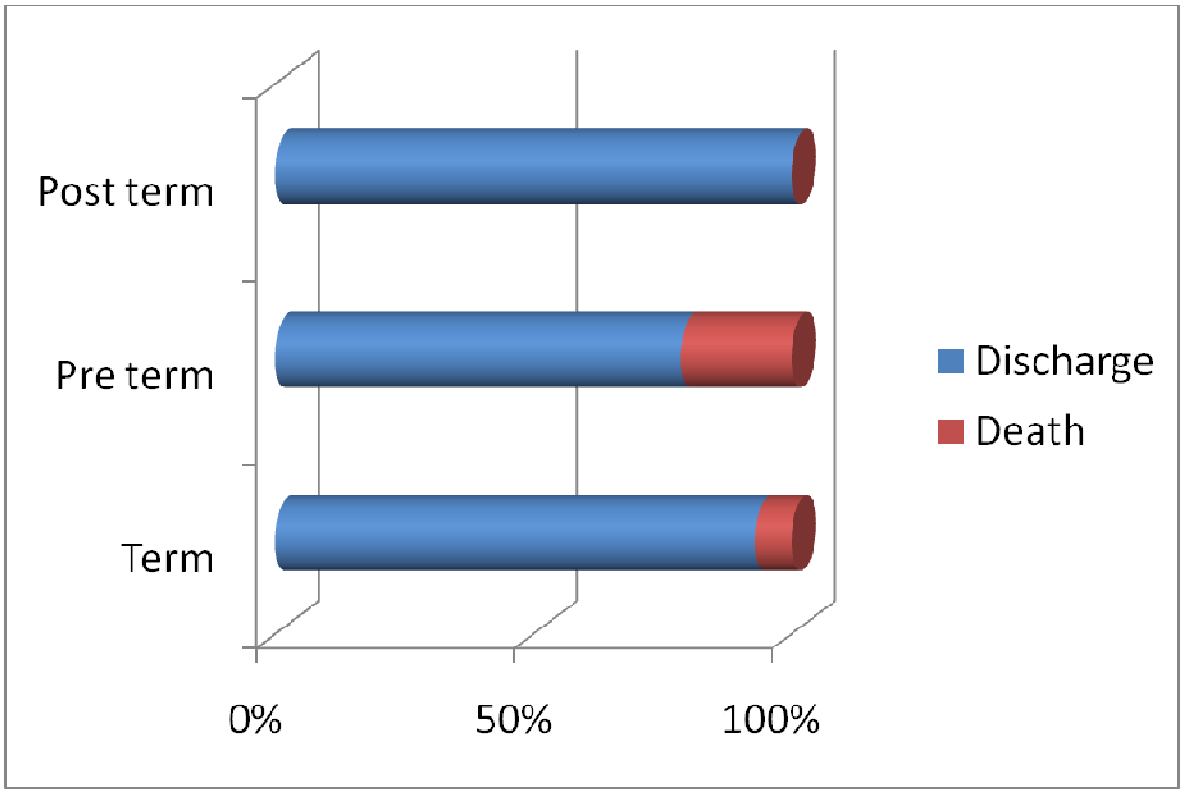
Of 132 babies 57 were term and 70 were preterm. Of term babies 28 (49.1%) were SGA & 19 (33.3%) were AGA. Of preterm babies 38 (54.3%) were SGS & 27 (38.6%) were AGA.

INFLUENCE OF GESTATIONAL AGE AND OUTCOME

GESTATIONAL AGE	DISCHARGE	DEATH	TOTAL
TERM	53	4	57
Row %	93	7	100
Col %	46.9	21.1	43.2
PRETERM	55	**15**	70
Row %	78.6	21.4	100
Col %	48.7	**78.9**	53
POST TERM	5	0	5
Row %	100	0	100
Col %	4.4	0	3.8
TOTAL	113	19	132
Row %	85.6	14.4	100
Col %	100	100	100

Out of total 19 deaths 15 (78.9%) babies were delivered preterm and 4 (21.1%) were term which is **statistically significant (p<0.05).**

INFLUENCE OF GESTATIONAL AGE ON OUTCOME



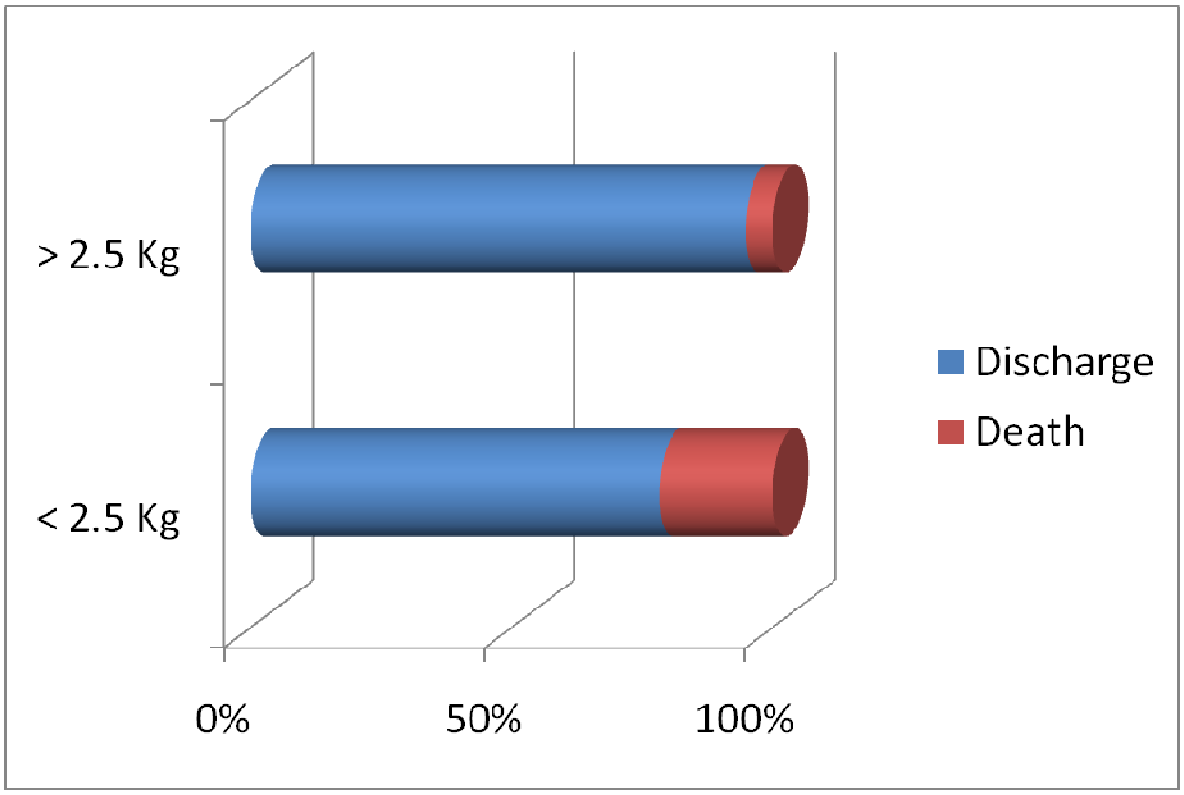
INFLUENCE OF BIRTH WEIGHT ON OUTCOME

B.WT	DISCHARGE	DEATH	TOTAL
<2.5 Kg	58	**16**	74
Row %	78.4	21.6	100
Col %	51.3	**84.2**	56.1
> 2.5 Kg	55	3	58
Row %	94.8	5.2	100
Col %	48.7	15.8	43.9
TOTAL	113	19	132
Row %	85.6	14.4	100
Col %	100	100	100

Of 19 deaths 16 (84.2%) were low birth weight and 3 (15.8%) were born of normal birth weight. Mortality was significantly higher in LBW babies.

Chi-square un corrected 2-tailed p – 0.00754, chi-square Mantel- Haenszel 2 tailed-p – 0.0077

INFLUENCE OF BIRTH WEIGHT ON OUTCOME

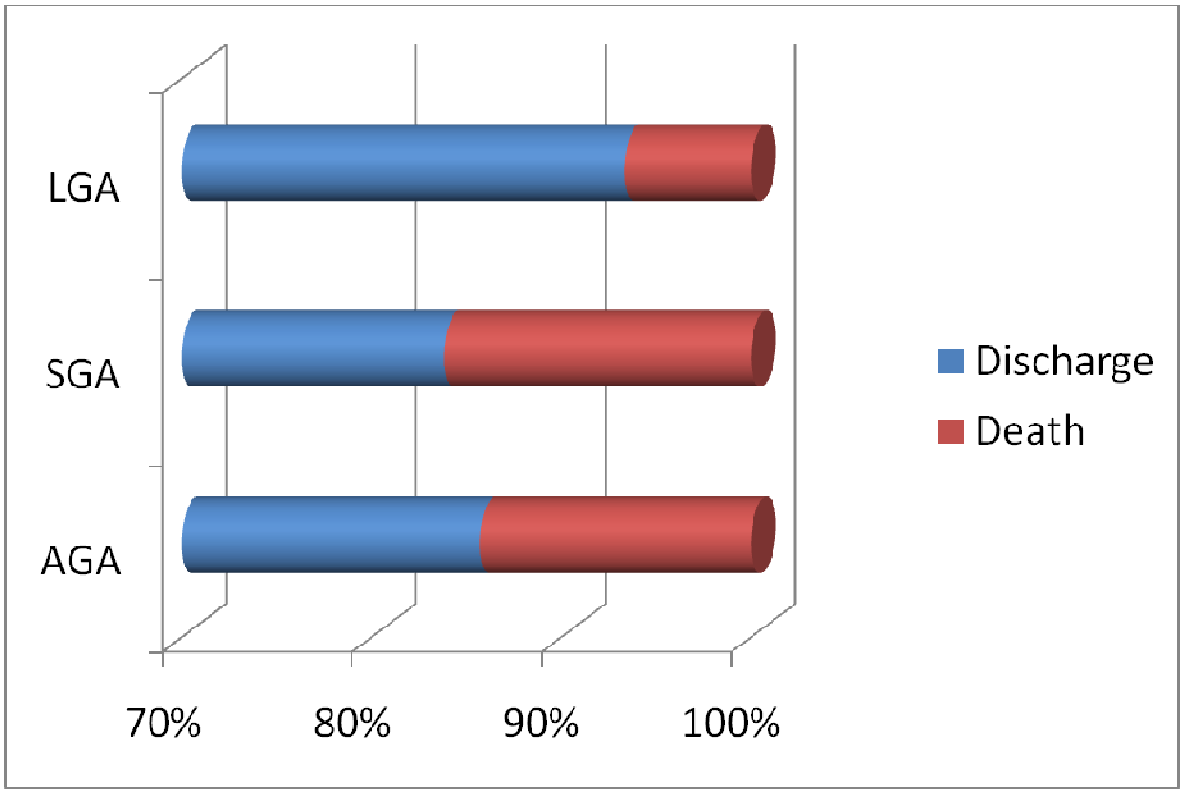


INTRA UTERINE NUTRITIONAL STATUS AND OUTCOME OF NEONATAL SEIZURES

NUTRITIONAL STATUS	DISCHARGE	DEATH	TOTAL
AGA	42	7	49
Row %	85.7	14.3	100
Col %	37.2	36.8	37.1
SGA	57	11	68
Row %	83.8	16.2	100
Col %	50.4	57.9	51.5
LGA	14	1	15
Row %	93.3	6.7	100
Col %	12.4	5.3	11.4
TOTAL	113	19	132
Row %	85.6	14.4	100
Col %	100	100	100

Out of 19 deaths 7 (36.8%) AGA, 11(57.9%) SGA, 1 (5.3%) LGA. Mortality was higher in SGA babies which was **statistically not significant (p – 0.638).**

INTRA UTERINE NUTRITIONAL STATUS AND OUTCOME OF NEONATAL SEIZURES



INCIDENCE

The incidence of neonatal seizures found in our study was 11.7/1000 live births, which is similar to studies conducted by Ajay kumar et al [11] (11.7/1000), shah GS et al [34] (10.3/1000), Amar et al [33] (16.69/1000) live births. Bergman et al (1983) [24] and Eriksson et al (1979) [30] reported incidence rates of seizures range from 1.5 to 5.5 in 1000 neonates. Cloherty et al [8] stated that in earlier reports, seizures occurred in up to 3 in 1,000 full-term infants and up to 60 in 1,000 premature infants. However, the reported incidence of neonatal seizures varies widely across studies, a variability that is primarily the result of inconsistent diagnostic criteria, as well as the often subtle clinical manifestations of neonatal seizures, and their potential confusion with non epileptic neonatal behaviors.

SEX DISTRIBUTION

It showed preponderance towards male babies of 57.5%, while females contributing with 42.5% with male to female ratio of 1.4: 1 in our study. This is comparable to Shah GS et al [34], Ajay et al [11], Cockburn et al, Fredrichsen et al and Mc.Intyre et al all showed male preponderance. Sanjeev kumar digra et al [40] (Jammu, India) reported male: female ratio of 2.4: 1 and explained it was because of increased care given to male sex while female babies were denied of medical care when it was needed.

GESTATIONAL AGE

Of 132 neonatal seizures, 43.2% were term and 53% were preterm, which was about 0.58% of all live term babies and 5.18% of all live preterm babies respectively. This is similar to Ajay et al [11], who reported as incidence in term babies was 0.69% and 6.14% in preterm. Rennie JM et al [16], Bernes and Kaplan [15] in PCNA (1990) and Laroia et al [14] reported varying incidence in term from 0.1 to 0.5% and 10 – 22.7% in preterm which is attributed due to the involvement of multiple factors like maternal medical illness, socio-economic status, health facilities available etc.

Meharban Singh [42] reported incidence of 0.5% – 0.8% in term babies and 6-12% in babies weighing <1500g.

INTRA UTERINE NUTRITIONAL STATUS

In our study, 51.5% babies were SGA, 37.1% AGA. The occurrence of neonatal seizures is more in SGA than AGA, which is similar to Ajay et al [11] (52.2%). Though equal number of deaths have occurred in both groups, SGA babies are more likely to go for complications because of underlying metabolic disturbances like hypoglycemia, hypocalcaemia, hypothermia etc.

BIRTH WEIGHT

In our study, 57.5% were low birth weight babies and 42.5% were born of normal weight, which is 2.9% of all LBW babies and 0.7% of babies weighing >2.5 Kg respectively. Kumar et al [11] reported 11.65% in LBW and 0.59% in normal birth weight babies. Shah GS [34] reported the incidence of neonatal seizures was 2 times higher in LBW babies. Lanska et al (1995) [17] reported seizure occurrence to be greatest in preterm or LBW babies compared with babies born at term. They found an incidence of seizures in all neonates to be 3.5 in 1000 but 57.5 in 1000 in VLBW (<1500g), 4.4 in 1000 LBW (1500-2499) and 2.8 in 1000 in normal birth weight neonates. Similarly, Kohelet and colleagues (2004) found an overall incidence of seizures in a cohort of VLBW infants to be 5.6%.

MORTALITY

19 (14.4%) babies died in our study, which is similar to Shah GS et al who reported 15% in his study. Ajay et al [11] reported 10% deaths, Tinuade ogunlesi et al [38] – 43.6% and Andre M et al [41] reported 22% mortality. Harris et al [35] in Australian pediatric journal 1998 reported mortality to be around 31% and stated that the leading risk factors were prematurity, LBW and severe birth asphyxia. In our study also, the mortality is significantly higher in preterm and LBW babies.

MODE OF DELIVERIES

In our study it was found that more incidence of seizures in breech and forceps deliveries. Similarly, Maheswari et al., [36] AIIMS, New Delhi in her study found that more number of babies delivered by forceps develop seizures when compared to normal deliveries. Pradhan et al., [37] Birmingham UK, reported that vaginal breech and emergency LSCS babies were significantly more likely to have low 5 min Apgar score require admissions in NICU and showed increased susceptibility towards birth trauma, birth asphyxia, neonatal seizures and death.

ETIOLOGY OF SEIZURES

Birth asphyxia (53%) was the most common cause of neonatal seizures in our study, similar to the previous studies as said above. This is because ours is a tertiary centre and cases were referred with improper antenatal care, untreated or partially treated PIH, ante partum hemorrhage, varying presentations of baby, fetal distress with meconeum stained liquor, undue prolongation of stages of labour.

Birth asphyxia	
Our study	**53%**
Shah GS et al [34]	44.44%
Kumar A et al [11]	45.7%
Amar et al [33]	42.7%
Sood A et al [9]	45%
cloherty [8]	45%
Levene et al & Goldherg et al [20]	15 – 53%

Sepsis, the next common cause of neonatal convulsions in our study similar to studies by Shah GS et al (20%) and Ledigo et al (17%). Frequent P/V examinations, prolonged premature rupture of membranes, poor hygiene of mother and family members contribute to increased sepsis.

Sepsis	
Our study	33.33%
Shah GS et al [34]	20%
Ledigo et al [32]	17%
cloherty [8]	5%

	Hypoglycemia	Hypocalcaemia
Our study	26.5%	18.2%
Shah GS et al [34]	20%	11%
Ajay et al [11]	23.33%	
Bergmen I et al[24] & Westerlaine [25]		1.1 – 22%
Cloherty [8]	5%	
Meharban singh [42]		10%

Hypoglycemia (26.5%) and hypocalcaemia (18.2%) were the metabolic disturbances observed in our study, which is similar to Shah GS et al, Ajay et al, Bergmen I et al and Westerlaine.

Intra cranial hemorrhage constitutes about 2.3% of babies in our study, which was lesser than that reported in Cloherty of 10%.

Causes remain unknown in 3% of cases, due to short life span of < 24 hrs and difficulty in shifting the ill child for ultrasound and CT scan.

Un identified cause	
Our study	3.9%
Shah GS et al [34]	10%
Ajay et al [11]	14%
Mizrahi EM & kellaway P et al [3, 18]	2.4 – 5.3%
Wasterlaine CGN et al [25]	
Meharban singh [42]	10%
cloherty [8]	10%

TYPES OF SEIZURES

	Subtle %	Multi focal %	Focal clonic %	Tonic %	Myoclonic %
Our study	40.9	35.6	13.6	9.1	0.8
Shah GS et al [34]	42.22	11.11	33.33	11.11	4.4
Ajay et al [11]	8.91	42.24	6.4	--	0.86
Domenech et al	42	33.9	64.3	10.7	16.1
Mizrahi & kellaway [18], Scher et al [2] Subtle seizures – Most common.					
Cloherty [8] & Meharban Singh [42] Over 50% - subtle seizures					

Subtle seizures (40.9%) were the commonest of all types followed by multifocal seizures (35.6%). This is similar to Shah GS et al, Mizrahi & Kellaway, Scher et al, Cloherty and Meharban Singh reporting subtle seizures are the commonest type accounting for over 50% of seizures. Ajay et al reported multifocal seizures as commonest of all types. Subtle seizures were common in preterm babies and multifocal seizures were common in term babies, both are statistically significant.

TIME OF ONSET OF SEIZURES

39.4% of babies developed seizures in < 24 hrs, 48.5% in 24-72 hrs and 12.1% in > 72 hrs. Sanjeev et al reported seizures occurring more in < 24 hrs. However 87.9% of cases had seizures in < 72 hrs of life. This is similar to Ajay et al & Shah GS et al. Multifocal seizures found to be common in < 24 hrs of life while subtle seizures were common in 24-72 hrs of life, which is statistically significant.

	< 24 hrs	**24 – 72 hrs**	**> 72 hrs**
Our study	39.4%	48.5%	12.1%
Sanjeev et al [40]	45.09%	25.49%	20.6
Ajay et al [11]	< 48 hrs – 57.8%		
Shah GS et al [34]	< 48 hrs – 85.5%		

SUMMARY

1) Incidence of neonatal seizures was 11.7 / 1000 live births.
2) Incidence was more in preterm than term neonates (5.18% Vs 0.58%)
3) Neonatal seizures constitute about 4.4% of all NICU admissions, among which preterm more than term (13.3% Vs 2.4%)
4) Neonatal seizures showed increased distribution in males than females (57.5% Vs 42.5%) with male to female ratio of 1.4: 1.
5) Neonatal seizures were more common in LBW babies (2.9%)
6) Neonatal seizures were more common in SGA babies than AGA babies (51.5 % Vs 37.5 %). Of all SGA neonatal seizures were more in preterm than term (55.9% Vs 41.2%)
7) Equal distribution of neonatal seizures in normal vaginal and LSCS deliveries (1.1%) with higher incidence among breech and forceps deliveries (2.6%)
8) Incidence of neonatal seizures was more than twice in twins as singleton deliveries (2.4% Vs 1.2%)
9) Birth asphyxia (53%) was the most common cause of all neonatal seizures followed by sepsis (33.3%), metabolic abnormalities, intra cranial hemorrhage etc.
10) Cause remains undetermined in 3.9% of cases

11) Subtle seizures (40.9%) were the commonest type of seizure observed followed by multifocal clonic (35.6%)

12) Subtle seizures were more common in preterm neonates while multifocal seizures were more common in term neonates.

13) Multifocal seizures were 2 times more common in < 24 hrs of life while subtle seizures common in 24 – 72 hrs.

14) 87.9% of neonatal seizures occurred in <72 hrs with 39.4% in < 24 hrs & 48.5% in 24-72 hrs.

15) Of the preterm neonates, neonatal seizures were more common in 24 – 72 hrs of life while in term neonates more common in < 24 hrs of life.

16) 19 neonates (14.4%) died of 132 cases and 113 were discharged from NICU.

17) Mortality was higher in LBW babies than babies of normal birth weight. (21.6% Vs 5.2%)

18) Mortality was higher in preterm neonates than term neonates (21.4% Vs 7%)

19) There was no difference in the outcome of neonatal seizures with intra uterine nutritional status.

CONCLUSION

1. Incidence of neonatal seizures was 11.7 / 1000 live births.
2. Neonatal seizures were more common in preterm, LBW and twin deliveries. Also higher among those delivered by breech and forceps deliveries.
3. 87.9% of neonatal seizures occurred within 72 hours of life.
4. Birth asphyxia was the most common cause of all neonatal seizures followed by sepsis.
5. Subtle seizures were the commonest type of seizure observed followed by multifocal clonic.
6. Subtle seizures were more common in preterm neonates and in 24 – 72 hours of life, while multifocal seizures were more common in term neonates and in < 24 hours of life.
7. Mortality was higher in LBW and preterm babies. There was no difference in the immediate outcome of neonatal seizures with intra uterine nutritional status.

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PROFORMA

- Name: Sex:
- Time of onset (hrs) : < 24 / 24 – 72 / > 72
- Type of seizures

  I. Subtle

  II. Focal clonic

  III. Multifocal

  IV. Tonic

  V. Myoclonic

Family H/O seizures

ANTENATAL HISTORY

- H/O fever with rashes : Yes / No
- H/O Pre ecclampsia : Yes / No
- H/O bleeding P/V : Yes / No
- H/O draining P/V for > 18 hrs : Yes / No
- H/O foul smelling liquor : Yes / No
- H/O maternal fever during labour /

< 2 wks before delivery : Yes / No

- H/O >3 P/V examinations during labour

  : Yes / No

- Any other history

BIRTH HISTORY

- Mode of delivery : Normal vaginal / breech /forceps / LSCS
- Baby cried soon after birth : Yes / No
- If no, when : after stimulation / resuscitation with BMV / ET tube / not cried at all
- Apgar score : 1 min ___ : 5 min ___
- Gestational age : term / preterm / post term (____ weeks)
- Birth weight : ___(Kg)

EXAMINATION

- Congenital anomalies
- External birth injuries
- Cry, activity, color
- Meconeum stained : Yes / No
- CVS, RS, P/A systemic examination

- CNS : Anterior fontanel : normal / bulging.

  Tone: N / increased / decreased.

  Reflexes : N /depressed / nil.

INVESTIGATIONS

- Hb
- PCV
- TC
- DC
- Platelets
- CRP
- Blood glucose
- Serum calcium
- Cerebrospinal fluid analysis
- NEC
- USG cranium
- EEG
- Urine for metabolic screening
- Serum magnesium
- CT scan

MASTER CHART

Sl. No.	Sex	Time of onset	Types	Gestati-onal age	Intra - uterine growth	Mode of delivery	Birth weight	Diagn-osis	Out come
1	1	1	1	1	1	3	2	1	1
2	1	2	1	1	2	4	2	1	2
3	2	1	1	1	1	1	2	2	1
4	2	1	1	1	1	1	2	1	1
5	2	2	1	1	1	3	2	7	1
6	1	1	1	1	2	1	1	8	1
7	1	1	1	1	1	4	2	2	1
8	2	2	1	1	2	3	1	7	1
9	2	1	1	1	1	1	2	1	1
10	1	2	1	1	2	3	1	7	1
11	1	1	1	1	1	1	2	6	1
12	1	2	1	1	3	4	2	14	2
13	2	2	1	1	1	1	2	7	1
14	1	2	1	1	2	1	2	1	1
15	1	2	1	1	1	3	2	1	1
16	1	2	1	1	3	1	2	1	1
17	2	2	1	1	1	1	2	7	1
18	1	1	2	1	1	1	2	5	1
19	1	1	2	1	1	3	2	8	1
20	2	2	2	1	1	3	2	7	1
21	1	2	2	1	1	1	2	5	1
22	1	2	2	1	1	1	2	1	1
23	2	2	2	1	1	3	2	7	1
24	1	2	2	1	1	1	2	1	1
25	1	2	2	1	2	3	1	3	1
26	2	1	3	1	1	1	2	5	1
27	1	1	3	1	2	1	1	3	1
28	2	2	3	1	3	1	2	1	1
29	1	1	3	1	1	3	2	11	1
30	2	2	3	1	2	1	1	3	1
31	2	1	3	1	3	4	2	1	1
32	1	2	3	1	2	1	1	1	1
33	2	1	3	1	2	3	1	1	1
34	2	1	3	1	2	3	1	1	1
35	1	2	3	1	3	1	2	1	1
36	1	1	3	1	2	1	1	3	1
37	2	2	3	1	3	1	2	4	1
38	2	1	3	1	2	3	1	2	1

39	1	1	3	1	2	1	1	4	1
40	2	1	3	1	2	3	1	2	1
41	2	1	3	1	3	1	2	3	1
42	2	2	3	1	2	4	1	6	1
43	1	1	3	1	2	1	1	4	1
44	2	2	3	1	3	3	2	10	1
45	1	2	3	1	2	4	1	5	2
46	2	1	3	1	2	1	1	2	1
47	1	3	3	1	2	1	1	7	1
48	2	1	3	1	2	3	2	1	1
49	2	3	3	1	3	1	2	3	1
50	1	1	3	1	2	1	2	1	1
51	2	1	3	1	2	3	2	8	1
52	1	1	3	1	2	3	2	5	1
53	2	1	3	1	2	3	2	5	1
54	2	1	3	1	2	1	2	6	1
55	1	1	3	1	3	1	2	3	1
56	1	1	3	1	2	2	2	14	2
57	2	1	3	1	2	1	2	6	1
58	2	1	1	2	2	3	1	1	1
59	2	2	1	2	3	3	2	1	1
60	2	2	1	2	1	3	1	1	2
61	2	2	1	2	2	3	1	1	1
62	2	1	1	2	1	4	1	2	2
63	1	2	1	2	2	1	1	1	1
64	1	3	1	2	1	3	1	3	1
65	2	1	1	2	2	1	1	2	2
66	2	2	1	2	1	2	1	2	1
67	1	3	1	2	1	3	1	5	1
68	2	2	1	2	2	1	1	2	1
69	1	1	1	2	1	4	1	2	2
70	2	2	1	2	2	1	1	1	1
71	2	3	1	2	1	3	1	6	1
72	1	2	1	2	2	2	1	2	1
73	2	1	1	2	1	1	1	10	1
74	1	2	1	2	1	1	2	1	1
75	1	2	1	2	1	3	2	13	1
76	2	3	1	2	2	1	1	3	1
77	1	1	1	2	2	3	1	1	2
78	1	2	1	2	1	1	2	1	1
79	2	3	1	2	2	3	1	6	1
80	2	2	1	2	1	3	2	6	1
81	1	1	1	2	2	2	1	7	1
82	2	2	1	2	1	1	1	1	1
83	1	3	1	2	2	1	1	1	2

84	1	1	1	2	2	1	1	3	1
85	2	1	1	2	1	3	2	1	1
86	2	2	1	2	2	1	1	1	1
87	1	2	1	2	2	3	1	3	1
88	1	2	1	2	1	2	2	6	1
89	2	2	1	2	2	1	1	1	1
90	2	2	1	2	2	1	1	4	1
91	1	2	1	2	1	3	1	8	2
92	1	2	1	2	2	1	1	1	1
93	2	1	2	2	1	1	1	7	1
94	2	2	2	2	2	3	1	2	2
95	1	2	2	2	1	3	1	10	1
96	2	1	2	2	1	1	1	3	1
97	2	2	2	2	1	1	1	12	2
98	1	2	2	2	1	1	1	13	2
99	1	3	2	2	2	2	1	2	1
100	2	3	2	2	1	1	1	4	1
101	1	3	2	2	2	3	1	2	1
102	2	1	3	2	1	2	1	2	1
103	1	1	3	2	2	1	1	4	1
104	1	2	3	2	1	3	1	5	2
105	1	1	3	2	2	3	1	2	1
106	2	2	3	2	2	1	1	2	1
107	2	1	3	2	1	1	1	2	1
108	1	2	3	2	2	1	1	5	2
109	1	2	3	2	1	3	1	9	1
110	2	1	3	2	2	1	2	5	1
111	1	2	3	2	1	1	1	5	1
112	2	2	3	2	2	3	2	5	1
113	2	1	3	2	2	3	1	7	2
114	1	3	3	2	2	1	1	8	1
115	1	3	4	2	2	3	1	6	1
116	1	1	4	2	2	1	1	10	1
117	2	1	4	2	2	3	1	5	2
118	1	2	4	2	2	4	2	13	1
119	1	2	4	2	3	3	2	5	1
120	1	2	4	2	3	4	2	14	1
121	2	2	4	2	2	1	1	6	1
122	1	2	4	2	2	3	2	13	1
123	1	2	4	2	3	1	2	3	1
124	2	2	4	2	3	1	2	8	1
125	2	3	4	2	2	3	1	8	2
126	2	3	4	2	2	3	2	5	1
127	1	1	5	2	2	1	1	7	1
128	1	2	3	3	1	1	1	2	1

129	2	3	3	3	1	1	1	2	1
130	2	2	2	3	2	1	2	1	1
131	1	1	1	3	1	3	2	2	1
132	2	2	1	3	2	3	2	5	1

KEY TO MASTER CHART

- Sex : 1. Male 2. Female
- Time of onset (hours) : 1. < 24 2. 24-72 3. >72
- Type of seizures : 1. Subtle
  2. Focal clonic
  3. Multifocal
  4. Tonic
  5. Myoclonic
- Mode of delivery : 1. Normal vaginal
  2. Breech
  3. Forceps
  4. LSCS
- Gestational age : 1. Term 2. Preterm 3. Post term
- Birth weight : 1. < 2.5 Kg. 2. > 2.5 Kg
- Intra uterine growth : 1. AGA, 2. SGA, 3. LGA

- Diagnosis: 1. Birth asphyxia
  2. Sepsis
  3. Hypoglycemia

4. Hypocalcaemia

5. Asphyxia / sepsis

6. Asphyxia / hypoglycemia

7. Asphyxia / hypocalcaemia

8. Sepsis / hypoglycemia

9. Sepsis / hypocalcaemia

10. Hypoglycemia / hypocalcaemia

11. Bilirubin encephalopathy

12. Anencephaly

13. Intracranial hemorrhage

14.Un known.

- Outcome: 1. Discharge. 2. Death.

பொற்கோர் தொடர்புக் படிவம்

ஆய்வுக் கூடம் : அரசு மருத்துவமனை,
சென்னை - 600 001.

நபர் கொடுத்த எண் :

நபர் கொடுத்த நாள் :

நபர் கொடுத்த இரண்டு (2) மாதிரிகள்.

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வில் பின்வரும் முடிவுகள் எனக்கு விளக்கப்பட்டது. அவற்றை நான் ஏற்றுக்கொள்கிறேன். அந்நோய் குறித்து விளக்கங்களை பொறுப்பாளர்களிடம் கேட்டுள்ளேன்.

எனது முழுமனதுடன் இந்த ஆய்வு செய்ய சம்மதிக்கிறேன். மற்ற மருத்துவர்களிடமோ மற்ற மருத்துவமனைகளிலோ மருத்துவம் செய்துகொள்ளும் முழுச்சுதந்திரம் இந்த ஆய்வில் இருந்து விலகி கொள்ளவும் என்னால் அறிந்துகொள்கிறேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதற்கு சம்பந்தமான வேறு ஏதேனும் பாதிப்புகளுக்காகவோ இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என் முழுமையான மருத்துவ ஆலோசனைகளை பெற்றதற்கு எனது ஒப்புதலை அளிக்கிறேன் என அறிந்து கொள்கிறேன். எனது முழுமனதை ஆய்வில் இருந்து விலகிக் கொள்ள முடிவு இது பொறுப்புள்ள என அறிகிறேன்.

இந்த ஆய்வின் முடிவு நிலை பற்றி தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை பிற தகவல் நாளிதழ்களையும் மருத்துவர் ஆய்வில் பங்குபெற்றுள்ள கொண்டால் அதை பிறகேற்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள தாமாக கொள்கிறேன். எனது கொடுப்பாளர் அறிவுரைகளின்படி நடந்து கொள்வதும் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவர் அனைத்து எனக்கான விளக்கங்களையும் தெளிவாக அளித்துள்ளார். என நடவடிக்கைகள் மற்றும் விதமான பாதிப்பு ஏற்பட்டாலும் அதிகாரத்தை மருத்துவர்களிடம் மாறான நோயாளருக்கு தெளிவாக எடுத்துரைத்தேன். மேலும் அதை பற்றுதலை அவர்களது நோயாளியாக என உறுதி அளிக்கிறேன்.

இந்த ஆய்வில் என் முழுமனதுடன் இருந்து. நிறுத்தி வைக்கிறேன், வரும் விளைவுகளுக்கு பாதிப்புகளையோ சொல்ல கொள்ள நான் முழு பொறுப்பாளர்களிடம் கூறியுள்ளேன்.

பொற்கோர் கையொப்பம் .....இடம்.....தேதி

கட்டைவிரல் ரேகை

பொற்றோரின் பெயர் மற்றும் விவரம் .....

ஆய்வாளரின் கையொப்பம்.....இடம்.....தேதி

ஆய்வாளரின் பெயர்.....

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-3

Title of the Work	: Neonatal Seizures - A Comprehensive Study
Principal Investigator	: Dr.J.Senthil Kumar
Designation	: PG in Paediatrics
Department	: Paediatrics

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 15.04.2010 at the Modernised Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

SECRETARY 18/5/10
IEC, SMC, CHENNAI

ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.